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RESPIRATORY CARE

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39TH YEAR—ESTABLISHED 1956

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- Clinical Practice Guidelines
  - Ventilator Circuit Changes
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  - Surfactant Replacement Therapy
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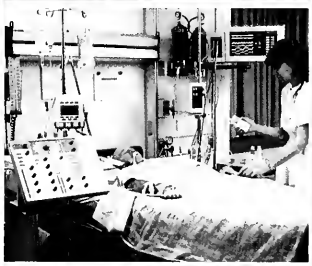

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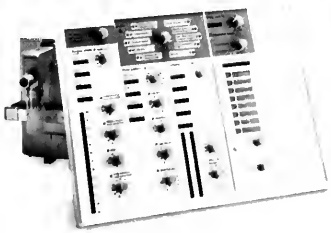

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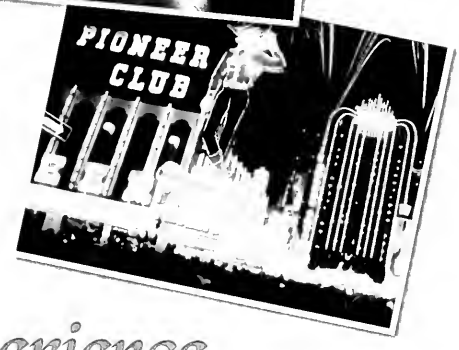

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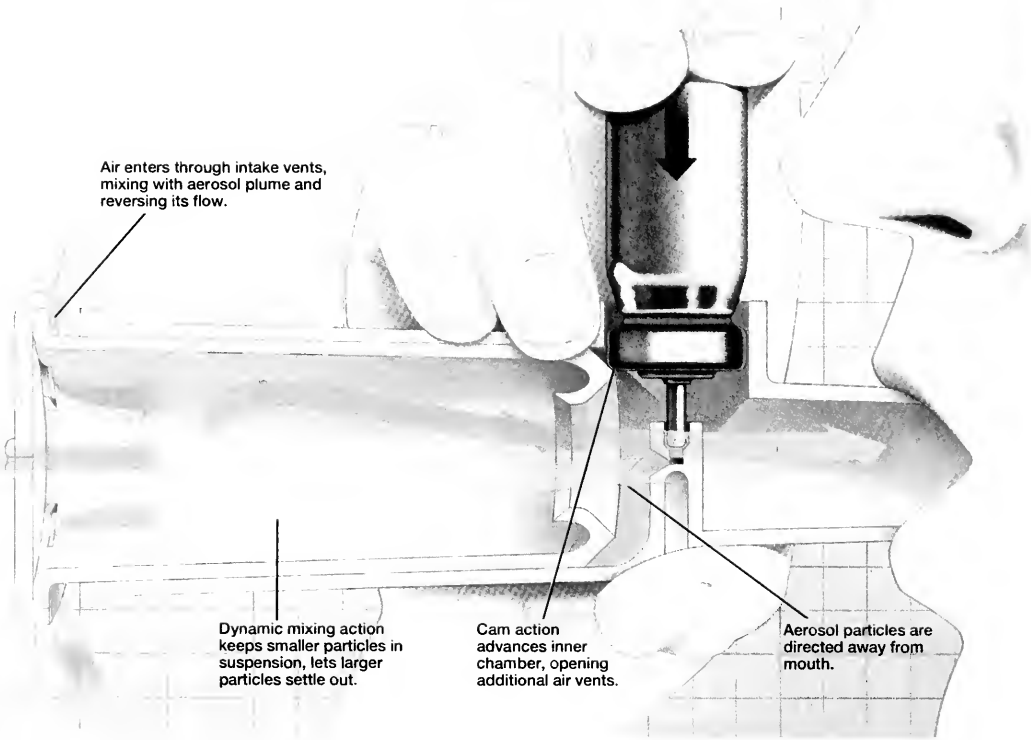

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The Letter or the Spirit: Consent for Research in CPR (editorial)—CM Olson. JAMA 1994;271(18):1445-1447. (Pertains to Lurie et al paper abstracted below.)

Blunted Perception and Death from Asthma (editorial)—PJ Barnes. N Engl J Med 1994;330(19):1383-1384. (Pertains to Kikuchi et al paper abstracted on Page 784.)

Experience with Pediatric Lung Transplantation—TM Egan. J Pediatr 1994;124(2):269. (Pertains to Noyes et al paper abstracted on Page 784.)

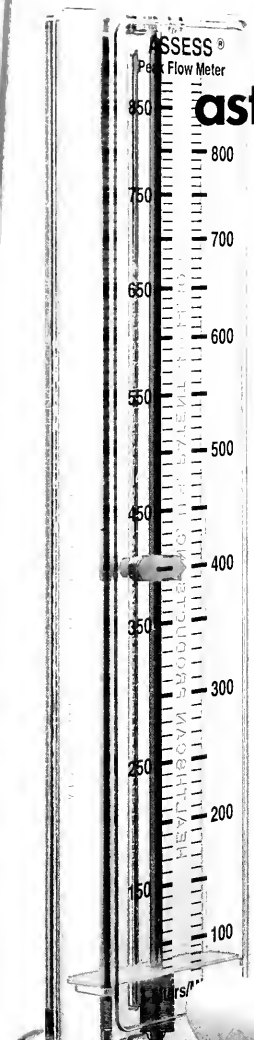
Nitric Oxide: Mediator, Murderer, and Medicine (review)—E Änggård. Lancet 1994;343(5):1199.

Salmeterol Xinafoate as Maintenance Therapy Compared with Albuterol in Patients with Asthma—GE D'Alonzo, RA Nathan, S Henochowicz, RJ Morris, P Ratner, SI Rennard. JAMA 1994;271(18):1412.

OBJECTIVE: To compare the efficacy and safety of inhaled salmeterol xinafoate, a long-acting β_2-adrenoceptor agonist, with that of albuterol, a short-acting inhaled β_2-agonist, in the treatment of asthma. DESIGN: Randomized, double-blind, placebo-controlled, parallel-group study. SETTING: Eleven outpatient clinical centers. SUBJECTS: A total of 322 male and female patients at least 12 years of age with chronic symptomatic asthma requiring daily therapy. INTERVENTION: Patients were treated with salmeterol xinafoate (42 μg inhaled twice daily), albuterol (180 μg inhaled four times daily), or placebo (four times a day) for 12 weeks; patients in all three groups could use inhaled albuterol as backup medication for breakthrough symptoms. MAIN OUTCOME MEASURES: Serial 12-hour forced expiratory flow in 1 second (FEV_1), peak expiratory flow (PEF), asthma symptoms, nocturnal awakenings due to asthma, episodes of asthma exacerbations, and electrocardiography. RESULTS: The mean area under the curve for FEV_1 throughout each 12-hour period was consistently greater after a single dose of salmeterol than after two doses of albuterol administered 6 hours apart ($p < 0.001$), with the difference ranging from 3.1 to 4.3 L · h. Salmeterol produced an average increase in morning and evening PEF of 26 and 29 L/min, respectively, over pretreatment values compared with decreases of −13 and −3 L/min, respectively, in the albuterol group and −2 L/min both in the morning and evening in the placebo group ($p < 0.001$). Patients in the salmeterol group had significantly fewer days and nights with symptoms than did either the albuterol or placebo group ($p < 0.001$). Responses to salmeterol were similar at Day 1 and at Week 12. Adverse events in all treatment groups were equally infrequent, and no clinically significant change in cardiac rhythm was observed with salmeterol treatment. CONCLUSION: Salmeterol inhaled twice daily is more effective than albuterol inhaled four times a day (or as needed) in patients with asthma requiring maintenance therapy. No deterioration of asthma control was observed with the use of salmeterol over a 3-month period.

Evaluation of Active Compression-Decompression CPR in Victims of Out-of-Hospital Cardiac Arrest—KG Lurie, JJ Shultz, ML Callaham, TM Schwab, T Gisch, T Rector, et al. JAMA 1994;271(18):1405.

OBJECTIVE: Active compression-decompression (ACD) cardiopulmonary resuscitation (CPR) appears to improve ventilation and coronary perfusion when compared with standard CPR. The objective was to evaluate potential benefits of this new CPR technique in patients with out-of-hospital cardiac arrest in St Paul, Minn. DESIGN: 10-month, prospective, randomized parallel-group design. SETTING: St Paul, Minn, population 270,000. PATIENTS: All normothermic victims of nontraumatic cardiac arrest older than 8 years who received CPR. MAIN OUTCOME MEASURES: Return of spontaneous circulation, admission to the intensive care unit (ICU), return of baseline neurological function (alert and oriented to person, place, and time), survival to hospital discharge, survival to hospital discharge with return of baseline neurological function, and complications. RESULTS: Seventy-seven patients received standard CPR and 53 patients received ACD CPR. The mean emergency medical services call response interval was less than 3.5 minutes. When all patients were considered, a higher percentage of ACD CPR patients had a return of spontaneous circulation and were admitted to the ICU vs standard CPR (45% vs 31%, and 40% vs 26%, respectively), but these trends were not statistically significant ($p < 0.10$ and $p < 0.10$). No statistically significant differences were found between hospital discharge rates (12 [23%] of 53 for ACD CPR vs 13 [17%] of 77 for standard CPR), return to baseline neurological function (10 [19%] of 53 for ACD CPR vs 13 [17%] of 77 for standard CPR), or return to baseline neurological function at hospital discharge (9 [17%] of 53 for ACD CPR vs 12 [16%] of 77 for standard CPR). Return of spontaneous circulation, ICU admission, and neurological recovery in both CPR groups were highly correlated with downtime (time from collapse to emergency medical system personnel arrival to the scene in witnessed arrests). With less than 10 minutes' downtime, survival to the ICU was 59% (19/32) with ACD CPR and 33% (16/49) with standard CPR ($p < 0.02$), return to baseline neurological function was 31% (10/32) with ACD CPR and 20% (10/49) with standard CPR ($p = 0.27$), and hospital discharge rate was 38% (12/32) with ACD CPR and 20% (10/49) with standard CPR ($p = 0.17$). Complication rates in patients admitted to the hospital were similar in both groups. CONCLUSIONS: This study demonstrates that ACD CPR appears to be more ef-



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fective than standard CPR in a well-defined subset of victims of out-of-hospital cardiac arrest during the critical early phases of resuscitation. Based on this study, a larger study should be performed to evaluate the potential long-term benefits of ACD CPR.

Artifactual Pulse-Oximetry Estimation in Neonates—HU Bucher, M Keel, M Wolf, K von Siebenthal, G Duc. Lancet 1994;343:1135.

Two sources of artifactual pulse-oximetry estimation were investigated in 20 neonates. Increased pressure on tissue due to inappropriate sensor fixation was mimicked with a blood pressure cuff. The error in arterial oxygen saturation (pSO_2) exceeded 2 SD (> 3%) in 25% subjects at 50 mm Hg which in an ancillary experiment was produced by 11 of 26 nurses fixing the sensor. Venous congestion at 30 and 40 mm Hg permitted normal detection of pulse rate but induced errors in pSO_2 over 2 SD in 15% and 30% of subjects, respectively. Pulse-oximeter values need to be scrutinized for these common errors.

Chemosensitivity and Perception of Dyspnea in Patients with a History of Near-Fatal Asthma—Y Kikuchi, S Okabe, G Tamura, W Hida, M Homma, K Shirato, T Takishima. N Engl J Med 1994;330(19):1329.

BACKGROUND: Many deaths from attacks of asthma may be preventable. However, the difficulty in preventing fatal attacks is that not all the pathophysiologic risk factors have been identified. METHODS: To examine whether dyspnea and chemosensitivity to hypoxia and hypercapnia are factors in fatal asthma attacks, we studied 11 patients with asthma who had had near-fatal attacks, 11 patients with asthma who had not had near-fatal attacks, and 16 normal subjects. Their respiratory responses to hypoxia and hypercapnia, determined by the standard rebreathing technique while the patients were in remission, were assessed in terms of the slopes of ventilation and airway occlusion pressure as a function of the percentage of arterial oxygen saturation and end-tidal carbon dioxide tension, respectively. The perception of dyspnea was scored on the Borg scale during breathing through inspiratory resistances ranging from 0 to 30.9 cm of water per liter per second. RESULTS: The mean (± SD) hypoxic ventilatory response (0.14 ± 0.12 liter per minute per percent of arterial oxygen saturation) and airway occlusion pressure (0.05 ± 0.05 cm of water per percent of arterial oxygen saturation) were significantly lower in the patients with near-fatal asthma than in the normal subjects (0.60 ± 0.35, $p < 0.001$, and 0.16 ± 0.08, $p < 0.001$, respectively) and the patients with asthma who had not had near-fatal attacks (0.46 ± 0.29, $p = 0.003$, and 0.15 ± 0.09, $p = 0.004$). The Borg score was also significantly lower in the patients with near-fatal asthma than in the normal subjects, and their lower hypoxic response was coupled with a blunted perception of dyspnea. CONCLUSIONS: Reduced chemosensitivity to hypoxia and blunted perception of dyspnea may predispose patients to fatal asthma attacks.

Inhaled Nitric Oxide in Infants Referred for Extracorporeal Membrane Oxygenation: Dose Response—NN Finer, PC Etches, B Kamstra, AJ Tierney, A Peliowski, CA Ryan. J Pediatr 1994;124:302.

To determine the role of inhaled nitric oxide (NO) in a population of critically ill hypoxic near-term infants and to determine the dose response to inhaled NO, we examined a consecutive group of 23 infants referred for neonatal extracorporeal membrane oxygenation (ECMO) who had an oxygen index of 20 or greater after treatment with bovine surfactant. Inhaled NO was administered in concentrations from 5 to 80 ppm in random order to 23 infants. Overall, 13 infants had a significant response (an improvement in arterial oxygen pressure > 10 mm Hg or arterial oxygen saturation > 10%) to the first administration of inhaled NO, and one infant had a late response. There was no significant difference in the response to inhaled NO as measured by changes in arterial oxygen pressure or in the alveolar-arterial difference in partial pressure of oxygen for any of the doses from 5 to 80 ppm. Thirteen infants had echocardiographic evidence of persistent pulmonary hypertension; 11 of these infants responded, compared with 3 responders among the 10 infants without persistent pulmonary hypertension of the newborn ($p < 0.01$). Overall, 11 infants required ECMO; there were two deaths in this group. Seven infants had congenital diaphragmatic hernia; five of those had a response to NO inhalation and four required ECMO. Our study demonstrates that there is no significant difference in response between low and high doses of inhaled NO and that this treatment may prevent the need for ECMO in some infants referred for this therapy, especially in infants with pulmonary hypertension. Prospective, controlled, randomized, and blinded trials of low doses of inhaled NO are needed to determine the clinical role of this potentially useful therapy.

Experience with Pediatric Lung Transplantation—BE Noyes, G Kurland, DM Orenstein, FJ Fricker, JM Armitage. J Pediatr 1994;124(2):261.

Heart-lung transplantation and lung transplantation have become accepted techniques in adult patients with end-stage cardiopulmonary disease. We report here our experience between July 1985 and March 1993 with 34 children (< 20 years) who underwent heart-lung (n = 18) or lung transplantation (n = 17). Indications for transplantation included cystic fibrosis (n = 9), congenital heart disease with Eisenmenger complex (n = 9), primary pulmonary hypertension (n = 8), pulmonary arteriovenous malformations (n = 2), desquamative interstitial pneumonia (n = 2), Proteus syndrome with multicystic pulmonary disease (n = 1), graft-versus-host disease (n = 1), rheumatoid lung disease (n = 1), and bronchiolitis obliterans and emphysema (n = 1). Twenty-six patients (76%) have survived from 1 to 88 months after transplantation; most patients have returned to an active lifestyle. Of the eight deaths, four were due to infections, two to multiorgan failure, one to posttransplant lymphoproliferative disease, and one to donor organ failure. Four of the patients who died had cystic fibrosis. Despite considerable morbidity related to infection, rejection, and function of the heart-lung and lung allograft in some patients our results with this potentially lifesaving procedure in the pediatric population have been encouraging.

Comparison of Manual and Mechanical Chest Percussion in Hospitalized Patients with Cystic Fibrosis—ML Bauer, J McDougal, RA Schoumacher. J Pediatr 1994;124(2):250.

We compared the efficacy of manual and mechanical chest percussion during hospitalization for acute exacerbations of cystic fibrosis by evaluating changes in spirometry values. Fifty-one participants were randomly assigned to receive manual or mechanical chest percussion three times a day. Twenty-two participated during one subsequent admission and were assigned to the opposite form of chest percussion. The two groups were equal in severity of illness (mean National Institutes of Health score [± SEM]: manual = 66.7 ± 2.2; mechanical = 65.8 ± 2.2; p = not significant). Mean improvement in forced expiratory volume at 1 second, forced vital capacity, and forced expiratory flow between 25% and 75% of forced vital capacity (± SEM) for manual percussion was 32.6% ± 7%, 27.2% ± 5%, and 38.1% ± 10%, and for mechanical percussion was 28.5% ± 4%, 28.7% ± 4%, and 25.1% ± 8%, respectively; p = not significant. Our participants did not prefer mechanical chest percussion. Although equal efficacy of outpatient therapy remains to be proved, this study suggests that patients can be encouraged to use the form of chest percussion that they prefer.

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ACLS INSTRUCTOR COURSE

TO BE OFFERED BEFORE AARC ANNUAL MEETING IN LAS VEGAS

On **Friday, December 9**, an American Heart Association **(AHA) Instructor Course** will be offered in conjunction with the AARC Annual Meeting. The course will be held at the Las Vegas Convention Center (site of all AARC Annual Meeting activities), from 7:30 AM to 6:00 PM Successful participants will be eligible to receive an ACLS instructor card from their local AHA chapter after teaching a course under the guidance of an experienced ACLS instructor. The course is being sponsored by the AARC Focus Group on Resuscitation in Acute Care Hospitals. Becoming an instructor will allow the successful individual to participate in teaching ACLS to respiratory care practitioners.

- Registrants need to be current ACLS providers and provide a recommendation from their last ACLS course director that they have potential to be an ACLS Instructor.
- Cost for the course is $175 per person. Deadline for registration is November 1, 1994.
- Minimum number of registrants is 18 (otherwise the course will be canceled); maximum is 36.
- Use registration form in the *Advance Program* for the AARC Annual Meeting which will mail in late summer to all AARC members. Register early to ensure your place in this unique course.

AMERICAN ASSOCIATION FOR RESPIRATORY CARE

The Effect of Directly Observed Therapy on the Rates of Drug Resistance and Relapse in Tuberculosis—SE Weis, PC Slocum, FX Blais, B King, M Nunn, GB Matney, et al. N Engl J Med 1994;330(17):1179.

BACKGROUND: Tuberculosis has reemerged as an important public health problem, and the frequency of drug resistance is increasing. A major reason for the development of resistant infections and relapse is poor compliance with medical regimens. In Tarrant County, Texas, we initiated a program of universal directly observed treatment for tuberculosis. We report the effect of the program on the rates of primary and acquired drug resistance and relapse among patients with tuberculosis. METHODS: We collected information on all patients with positive cultures for *Mycobacterium tuberculosis* in Tarrant County from January 1, 1980, through December 31, 1992. Through October 1986, patients received a traditional, unsupervised drug regimen. Beginning in November 1986, nearly all patients received therapy under direct observation by health care personnel. RESULTS: A total of 407 episodes in which patients received traditional treatment for tuberculosis (January 1980 through October 1986) were compared with 581 episodes in which therapy was directly observed (November 1986 through December 1992). Despite higher rates of intravenous drug use and homelessness and an increasing rate of tuberculosis during this 13-year period, the frequency of primary drug resistance decreased from 13.0% to 6.7% ($p < 0.001$) after the institution of direct observation of therapy, and the frequency of acquired resistance declined from 14.0% to 2.1% ($p < 0.001$). The relapse rate decreased from 20.9% to 5.5% ($p < 0.001$), and the number of relapses with multidrug-resistant organisms decreased from 25 to 5 ($p < 0.001$). CONCLUSIONS: The administration of therapy for *M tuberculosis* infection under direct observation leads to significant reductions in the frequency of primary drug resistance, acquired drug resistance, and relapse.

Trends in Cigarette Smoking among US Physicians and Nurses—DE Nelson, GA Giovino, SL Emont, R Brackbill, LL Cameron, J Peddicord, PD Mowery. JAMA 1994; 271(16):1273.

OBJECTIVE: To determine trends in cigarette smoking prevalence among physicians, registered nurses, and licensed practical nurses since 1974. DESIGN: Analyses of data on smoking prevalence among persons 20 years of age and older using combined National Health Interview Survey data sets from 1974, 1976, and 1977; 1978, 1979, and 1980; 1983 and 1985; 1987 and 1988; and 1990 and 1991. MAIN OUTCOME MEASURES: Prevalence of cigarette smoking and average annual change in smoking prevalence. RESULTS: Based on the data for 1990 and 1991, there were an estimated 18,000 physicians, 322,000 registered nurses, and 128,000 licensed practical nurses who smoked cigarettes in the United States. Compared with 1974, 1976, and 1977, by 1990 and 1991 cigarette smoking prevalence had declined from 18.8% to 3.3% among physicians (average annual decline of 1.15 percentage points); from 31.7% to 18.3% among registered nurses (average annual decline of 0.88 percentage point); and from 37.1% to 27.2% among licensed practical nurses (average annual decline of 0.62 percentage point). CONCLUSION: Since 1974, cigarette smoking has declined most rapidly among physicians, at an intermediate rate among registered nurses, and at a lower rate among licensed practical nurses. Because of their important roles as exemplars and health educators, persons in these occupations should not smoke.

Head Extension and Laryngeal View during Laryngoscopy with Cervical Spine Stabilization Maneuvers—RH Hastings, PR Wood. Anesthesiology 1994;80(4):825.

BACKGROUND: Direct laryngoscopy frequently is modified in patients with known or suspected cervical spine injury. The goals of this study were to measure the degrees of head extension required to expose the arytenoid cartilages and glottis if neck flexion were not possible and to determine whether in-line stabilization maneuvers alter the amount of head extension. METHODS: The subjects were anesthetized patients with normal cervical spines and Mallampati class 1 oropharyngeal views. Head extension was measured relative to a line drawn perpendicular to the table. Stabilization consisted of either passive immobilization, with the head held flat against a rigid board, or axial traction. RESULTS: Without stabilization, arytenoid cartilage exposure and the best view of the glottis was achieved with a $10 \pm 5°$ (mean ± SD) head extension and a $15 \pm 6°$ head extension, respectively ($n = 31$). Head immobilization reduced extension angles $4 \pm 5°$ for arytenoid exposure and $5 \pm 6°$ for best view compared with no stabilization. CONCLUSIONS: Head immobilization reduced head extension necessary for laryngoscopy. If head extension is construed to be potentially dangerous in patients with cervical spine injuries, head immobilization without traction might be the preferable stabilization technique.

Prolonged Inhalation of Low Concentrations of Nitric Oxide in Patients with Severe Adult Respiratory Distress Syndrome—LM Bigatello, WE Hurford, RM Kacmarek, JD Roberts, WM. Zapol. Anesthesiology 1994;80(4):761.

BACKGROUND: Nitric oxide (NO) inhalation selectively decreases pulmonary artery hypertension and improves arterial oxygenation in patients with the adult respiratory distress syndrome (ARDS). In this study of patients with severe ARDS, we sought to determine the effect of inhaled NO dose and time on pulmonary artery pressure and oxygen exchange and to determine which patients with ARDS are most likely to show this response. METHODS: Thirteen patients with severe ARDS (hospital mortality 67%) inhaled 0-40 parts per million (ppm) NO. Seven of these patients continued to breathe 2-20 ppm NO for 2-27 days. RESULTS: Inhaling 5-40 ppm NO decreased mean pulmonary artery pressure in a dose-related fashion (from 34 ± 7 to 30 ± 7 mmHg at 20 ppm NO). Systemic arterial pressure did not change. The ratio of arterial oxygen tension to inspired oxygen fraction increased (from 126 ± 36 to 149 ± 38 mmHg) and the venous admixture decreased (from 31.2 ± 5.5 to $28.2 \pm 5.2\%$) without a clear dose-response effect. During prolonged NO inhalation, 2-20 ppm NO effectively reduced mean pulmonary artery pressure (38 ± 7 vs 31 ± 6 mmHg) and increased arterial oxygen tension (79 ± 10 vs 114 ± 27 mmHg) without evidence of tachyphylaxis. The decrease of pulmonary vascular resistance during NO inhalation correlated with the level of pulmonary vascular resistance without NO ($r = -0.72$). The reduction of venous admixture correlated with the level of venous admixture without NO ($r = -0.78$). CONCLUSIONS: Long-term NO inhalation at low concentrations selectively decreases mean pulmonary artery pressure and improves arterial oxygen tension in patients with ARDS. The selective pulmonary vasodilation effect is most pronounced in ARDS patients with the greatest degree of pulmonary vasoconstriction.

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Blood Gas and Related Measurements: Laboratory versus Bedside Devices • July 26

Featuring Barry R. Shapiro, MD
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Presents the various means of obtaining blood gas information, invasive and noninvasive devices, the need for such devices, and when they should be used.

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Respiratory Care Practitioners Take Pride In What They Do

Celebrate RC Week this year by demonstrating your dedication to your profession. RCPs across the nation will join you in showing your co-workers, community, family, and local officials that you care about your patients, profession, and place in the health care community. Throughout the catalog are *RC Week Ideas* to show you how your fellow RCPs commemorate RC Week and how you can, too!

❶ T-shirt

Rich, jewel-tone colors of forest green, purple, and gold display your professional pride on a 50/50 cotton/polyester blend T-shirt. Medium, large, extra large, and extra-extra large available.

Item R16 $9.50 ($11.50 nonmembers)
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❷ Sweatshirt

Don't wait to order this classic RC Week sweatshirt in fashionable forest green. Sweatshirts are 50/50 blend. Medium, large, extra large, and extra-extra large available.

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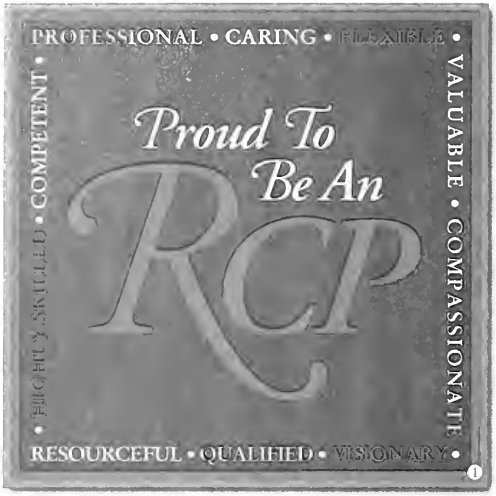
❸ RCP Cap

Add the finishing touch to your RC Week apparel with this forest green cap, silk-screened in metallic gold.

Item R17 $6 ($9 nonmembers)

Thanks to Barbara Hendon, BA, RRT, and AARC employees Robert Czachowski, PhD, and Lisa Best for modeling the RC Week apparel in this catalog.



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Display this 17" x 25" poster in your department or cafeteria to proclaim the respiratory care practitioner's key role on the health care team. Sold with RC Week dates before Oct. 1 and without the dates after that.

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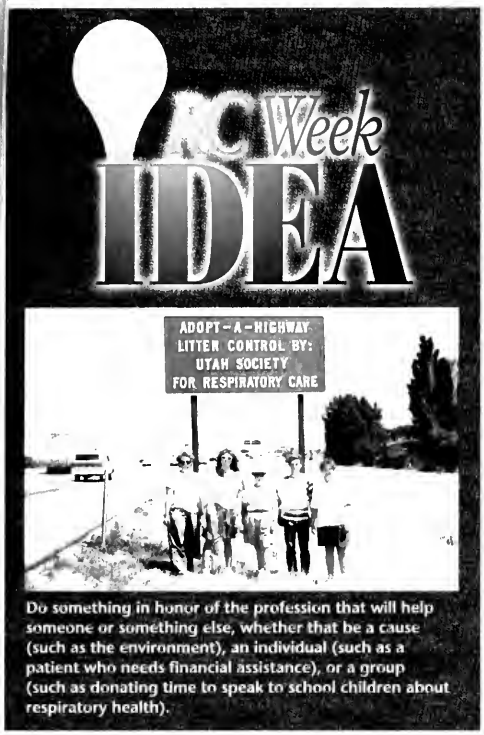
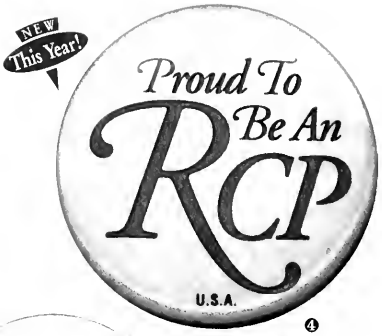


Do something in honor of the profession that will help someone or something else, whether that be a cause (such as the environment), an individual (such as a patient who needs financial assistance), or a group (such as donating time to speak to school children about respiratory health).



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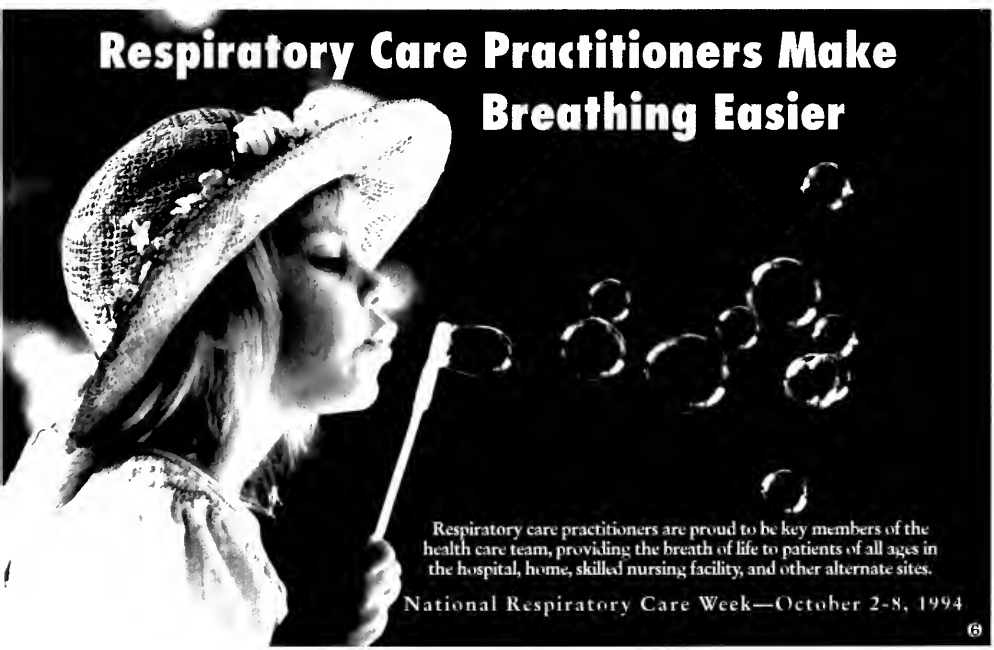

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INTRODUCING

A NEW SOLUTION IN NEBULIZATION

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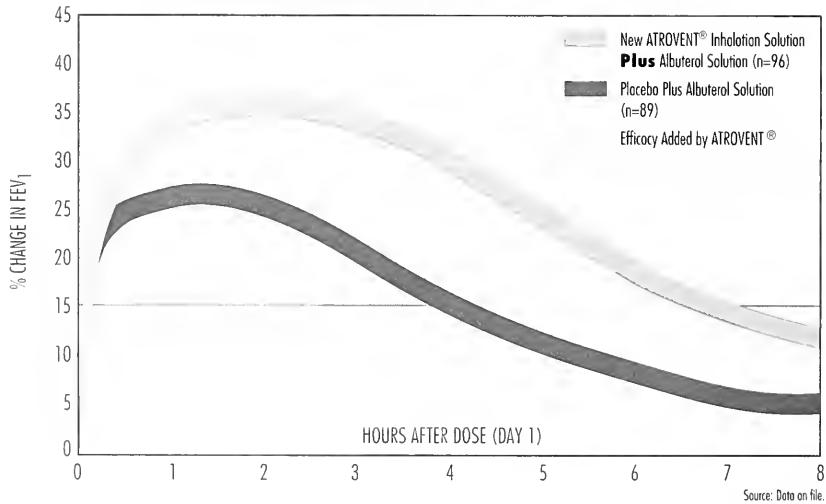
Significantly raises the efficacy curve

In combination with albuterol



Source: Data on file.

In patients with COPD, ATROVENT Inhalation Solution plus albuterol solution produced significantly greater improvement in FEV_1 than albuterol alone.[1*]

ATROVENT Inhalation Solution is a bronchodilator for the maintenance treatment of bronchospasm in COPD.

* Multicenter, randomized, double-blind, parallel trial conducted in 195 patients with COPD in whom pretreatment mean FEV_1 = 39% of predicted normal.

Please see brief summary of Prescribing Information on last page of this advertisement.

. . .without lowering the safety profile

The combination of ATROVENT Inhalation Solution and albuterol solution produced no clinically significant increase in side effects compared with albuterol alone.[2]

ATROVENT Inhalation Solution has a low incidence of systemic anticholinergic side effects.[3]

The use of ATROVENT Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation.

Combination of ATROVENT and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation.

ATROVENT should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy or bladder-neck obstruction.

Please see Prescribing Information for all adverse events, regardless of drug relationship, reported in the 12-week controlled trials of ATROVENT Inhalation Solution.

NEW

ATROVENT®

(ipratropium bromide) 500 mcg unit dose 0.02%

INHALATION SOLUTION

Added efficacy without added side effects*

*When used in combination with a beta-agonist solution, ATROVENT does not significantly increase side effects associated with the beta agonist alone.

NEW

ATROVENT

(ipratropium bromide) 500 mcg unit dose 0.02%

INHALATION SOLUTION

ADDED EFFICACY WITHOUT ADDED SIDE EFFECTS*

Atrovent®
(ipratropium bromide)
Inhalation Solution

Brief Summary of Prescribing Information

CONTRAINDICATIONS Atrovent® (ipratropium bromide) is contraindicated in known or suspected cases of hypersensitivity to ipratropium bromide, or to atropine and its derivatives

WARNINGS The use of Atrovent® (ipratropium bromide) Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation. Combination of Atrovent and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation

PRECAUTIONS General Atrovent® (ipratropium bromide) should be used with caution in patients with narrow angle glaucoma, prostatic hypertrophy or bladder-neck obstruction

Information for Patients Patients should be advised that temporary blurring of vision, precipitation or worsening of narrow-angle glaucoma or eye pain may result if the solution comes into direct contact with the eyes. Use of a nebulizer with mouthpiece rather than face mask may be preferable, to reduce the likelihood of the nebulizer solution reaching the eyes. Patients should be advised that Atrovent Inhalation Solution can be mixed in the nebulizer with albuterol if used within one hour. Compatibility data are not currently available with other drugs. Patients should be reminded that Atrovent Inhalation Solution should be used consistently as prescribed throughout the course of therapy.

Drug Interactions Atrovent has been shown to be a safe and effective bronchodilator when used in conjunction with beta adrenergic bronchodilators. Atrovent has also been used with other pulmonary medications, including methylxanthines and corticosteroids, without adverse drug interactions.

Carcinogenesis, Mutagenesis, Impairment of Fertility Two-year oral carcinogenicity studies in rats and mice have revealed no carcinogenic potential at dietary doses up to 6 mg/kg/day of Atrovent. Results of various mutagenicity studies (Ames test, mouse dominant lethal test, mouse micronucleus test and chromosome aberration of bone marrow in Chinese hamsters) were negative. Fertility of male or female rats at oral doses up to 50 mg/kg/day was unaffected by Atrovent administration. At doses above 90 mg/kg, increased resorption and decreased conception rates were observed.

Pregnancy *TERATOGENIC EFFECTS Pregnancy Category B* Oral reproduction studies performed in mice, rats and rabbits at doses of 10, 100, and 125 mg/kg respectively, and inhalation reproduction studies in rats and rabbits at doses of 1.5 and 1.8 mg/kg (or approximately 38 and 45 times the recommended human daily dose) respectively, have demonstrated no evidence of teratogenic effects as a result of Atrovent. However, no adequate or well-controlled studies have been conducted in pregnant women. Because animal reproduction studies are not always predictive of human response, Atrovent should be used during pregnancy only if clearly needed.

Nursing Mothers It is not known whether Atrovent is excreted in human milk. Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that Atrovent® (ipratropium bromide) would reach the infant to a significant extent, especially when taken by inhalation, since Atrovent is not well absorbed systemically after inhalation or oral administration. However, because many drugs are excreted in human milk, caution should be exercised when Atrovent is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children below the age of 12 have not been established.

ADVERSE REACTIONS Adverse reaction information concerning Atrovent® (ipratropium bromide) Inhalation Solution is derived from 12-week active-controlled clinical trials. Additional information is derived from foreign post-marketing experience and the published literature.

All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials appear in the table below.

All Adverse Events, from Double-blind, Parallel, 12-week Studies of Patients with COPD*

	PERCENT OF PATIENTS				
	Atrovent® (500 mcg t i d) n=219	Alupent® (15 mg t i d) n=212	Atrovent®/Alupent® (500 mcg t i d/ 15 mg t i d) n=108	Albuterol (2.5 mg t i d) n=205	Atrovent®/Albuterol (500 mcg t i d/ 2.5 mg t i d) n=100
Body as a Whole-General Disorders					
Headache	6.4	5.2	6.5	6.3	9.0
Pain	4.1	3.3	0.9	2.9	5.0
Influenza-like symptoms	3.7	4.7	6.5	0.5	1.0
Back pain	3.2	1.9	1.9	2.4	0.0
Chest pain	3.2	4.2	5.6	2.0	1.0
Cardiovascular Disorders					
Hypertension/ Hypertension Aggravated	0.9	1.9	0.9	1.5	4.0
Central & Peripheral Nervous System					
Dizziness	2.3	3.3	1.9	3.9	4.0
Insomnia	0.9	0.5	4.6	1.0	1.0
Tremor	0.9	7.1	8.3	1.0	0.0
Nervousness	0.5	4.7	6.5	1.0	1.0
Gastrointestinal System Disorders					
Mouth Dryness	3.2	0.0	1.9	2.0	3.0
Nausea	4.1	3.8	1.9	2.9	2.0
Constipation	0.9	0.0	3.7	1.0	1.0
Musculo-skeletal System Disorders					
Arthritis	0.9	1.4	0.9	0.5	3.0
Respiratory System Disorders (Lower)					
Coughing	4.6	8.0	6.5	5.4	6.0
Dyspnea	9.6	13.2	16.7	12.7	9.0
Bronchitis	14.6	24.5	15.7	16.6	20.0
Bronchospasm	2.3	2.8	4.6	5.4	5.0
Sputum Increased	1.4	1.4	4.6	3.4	0.0
Respiratory Disorder	0.0	6.1	6.5	2.0	4.0
Respiratory System Disorders (Upper)					
Upper Respiratory Tract Infection	13.2	11.3	9.3	12.2	16.0
Pharyngitis	3.7	4.2	5.6	2.9	4.0
Rhinitis	2.3	4.2	1.9	2.4	0.0
Sinusitis	2.3	2.8	0.9	5.4	4.0

*All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials

Additional adverse reactions reported in less than three percent of the patients treated with Atrovent include tachycardia, palpitations, eye pain, urinary retention, urinary tract infection and urticaria. A single case of anaphylaxis thought to be possibly related to Atrovent has been reported. Cases of precipitation or worsening of narrow-angle glaucoma and acute eye pain have been reported.

Lower respiratory adverse reactions (bronchitis, dyspnea and bronchospasm) were the most common events leading to discontinuation of Atrovent therapy in the 12-week trials. Headache, mouth dryness and aggravation of COPD symptoms are more common when the total daily dose of Atrovent equals or exceeds 2,000 mcg.

HOW SUPPLIED Atrovent® (ipratropium bromide) Inhalation Solution Unit-Dose Vial is supplied as a 0.02% clear, colorless solution containing 2.5 ml with 25 vials per foil pouch (NDC 0597-0080-62). Each vial is made from a low density polyethylene (LDPE) resin.

Consult package insert before prescribing

AS-BS-9/93

* When used in combination with a beta-agonist solution, ATROVENT does not significantly increase side effects associated with the beta agonist alone

References: 1. Levin DC, Little KS, Laughlin KR, Galbraith JM. Ipratropium bromide solution in COPD augments extent and duration of FEV_1 increases achieved by albuterol up to 8 hours. *Chest* 1993;104(suppl to No. 2):112S. Abstract. **2.** Data on file, Boehringer Ingelheim Pharmaceuticals, Inc. **3.** ATROVENT® (ipratropium bromide) Inhalation Solution full Prescribing Information

Boehringer Ingelheim

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AS-5381

The Effect of Time and Warming on Breathing Circuit Compliance

Kim L Valeri MS RRT, Thomas V Hill MS RRT, Arthur A Taft MHS RRT,
Shelley C Mishoe MEd RRT, and Cynthia J Phillips BS RRT

BACKGROUND: Some microprocessor-controlled ventilators correct the gas volume delivered to the patient to compensate for volume lost due to the compliance of the breathing circuit (C_{BC}). Because C_{BC} is usually calculated only when the circuit is initially placed in use, changes in C_{BC} that occur over time for various reasons may result in inaccurate correction of delivered tidal volume (V_T). The purpose of this study was to determine whether C_{BC} changed with time. MATERIALS & METHODS: We measured the compliance of 3 different types of breathing circuits (BCs) attached to a ventilator set at V_T = 1.0 L, f = 12, $\dot{V}_{max}$ = 60 L/min, and F_{IO_2} = 0.21. After an initial measurement of compliance, the BC were heated to 34°C inside an isolette, connected to a test lung, and ventilated for 48 hours. The compliance of the test lung was adjusted so that peak inspiratory pressure was 50 cm H_2O. Five samples of each BC were tested. Compressible volume was measured at 1, 3, 6, 12, 24, and 48 hours by injecting air from a calibration syringe into the BC (with all ports capped) until pressure reached 100 cm H_2O. The exhalation valve was opened and the released volume was measured with a calibration analyzer. The study was then repeated with the circuits maintained at room temperature. Data were analyzed using analysis of variance and Tukey's honestly significant differences tests with the level of significance set at 0.05. RESULTS: There was no significant difference in C_{BC} of the Hudson ethylene vinyl acetate circuit (mean [SD] 1.50 [0.08] mL/cm H_2O, p = 0.26) and Puritan-Bennett polyethylene circuit (1.47 [0.06] mL/cm H_2O, p = 0.24) over 48 hours. C_{BC} increased from 2.31 (0.06) mL/cm H_2O to 2.52 (0.06) mL/cm H_2O during the first hour in the Hudson kraton circuit, and the difference was significant (p < 0.001, 1 hour vs time zero); there was no significant difference after the 1 hour measurement (p = 0.67, 1 hour vs all subsequent time periods). There was no significant difference in calibration of the unheated circuits. CONCLUSION: Although we found a statistically significant increase in the compliance of the Hudson kraton circuit, there were no clinically important changes in the compliance of any of the breathing circuits over a 48-hour period. It may not be necessary to recalculate breathing circuit compliance for the purpose of correcting V_T delivered to the patient after the initial setup. [Respir Care 1994;39(8):793-796.]

Ms Valeri is Instructor, Respiratory Care Program, Sinclair Community College, Dayton, Ohio; Mr Hill is Professor and Chairman, Respiratory Care Program, Kettering College of Medical Arts, Kettering, Ohio; Mr Taft is Associate Professor and Director of Clinical Education, Ms Mishoe is Associate Professor and Chair, and Ms Phillips is Instructor—Department of Respiratory Therapy, School of Allied Health Sciences, Medical College of Georgia, Augusta, Georgia.

A version of this paper was presented during the RESPIRATORY CARE OPEN FORUM at the AARC Annual Meeting held in San Antonio, Texas, December 12-15, 1992.

None of the authors has a financial interest in any of the products mentioned in this paper.

Reprints: Thomas V Hill MS RRT, Respiratory Care Program, College of Medical Arts, 3737 Southern Boulevard, Kettering OH 45429.

Background

During mechanical ventilation, the tidal volume (V_T) delivered to the patient is monitored closely and attempts are made to minimize variation in its value. Other investigators have reported that a portion of the V_T set on a mechanical ventilator never reaches the patient but remains in the ventilator, humidifer, and inspiratory circuit due to compressed volume.[1-6] One of the reasons given for this compressed volume is the compliance of the breathing circuit.[2] This loss of volume may be clinically important and may require adjustments in the V_T set on the ventilator to compensate for its loss. Campbell et al studied the effects of temperature on breathing circuit compliance and reported that C_{BC} increased with temperature.[3]

Ventilators incorporating microprocessor controls, which are able to compensate for the volume lost due to the compliance of the breathing circuit (C_{BC}) have become more common in respiratory care in recent years. Breathing circuit compliance is generally only calculated when the circuit is initially placed in use such as during the extended self test (EST) used by the Puritan-Bennett 7200 series.* On the 7200 series, the pneumatic system corrects each mandatory breath for the compliance of the breathing circuit. The compliance volume is calculated by multiplying the C_{BC} (calculated during the EST) by the average peak inspiratory pressure (PIP) of the last four mandatory breaths. This compliance volume is then added to the V_T set and this corrected V_T is delivered on the subsequent breath.[7] The BEAR-5 ventilator allows the practitioner to enter a circuit compliance factor to be used by the ventilator to automatically correct the V_T so that the set V_T is actually delivered to the patient.[8] However, if breathing circuit compliance changes during the time the circuit is in use, and compliance has not been recalculated, the microprocessor may make an inaccurate adjustment to set V_T. None of the studies we identified measured changes in C_{BC} over time. Because changes in C_{BC} may result in changes in delivered V_T, we investigated the effect of time on C_{BC}. Because breathing circuits are generally heated during clinical use, we investigated the separate effects of time and warming on C_{BC}.

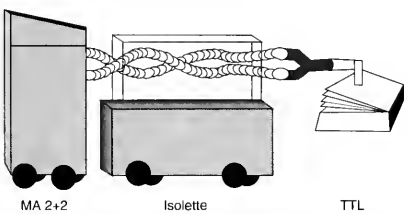

Fig. 1. Diagram showing the breathing circuit lying inside the incubator heated to 34°C. It is connected to the ventilator (MA 2+2) and lung simulator (TTL).

Materials and Methods

We measured the compliance of three different types of disposable breathing circuits of equal length (Hudson ethylene vinyl acetate [EVA], Hudson kraton [HK], and Puritan-Bennett polyethylene [PBP]). Five circuits of each type were tested. Circuits were attached to a Bennett MA2+2 ventilator with the humidifier by-passed and to a lung simulator (Fig. 1). An initial compliance measurement was taken and circuits were then heated to 34°C inside an infant incubator for 48 hours. The ventilator was set with V_T = 1.0 L, f = 12, $\dot{V}_{max}$ = 60 L/min, F_{IO_2} = 0.21. The compliance of the lung simulator was adjusted to maintain a peak inspiratory pressure of 50 cm H_2O.

The ventilator's manometer and V_T control setting were calibrated prior to each testing session using a calibration analyzer. Compliance was measured initially and at 1, 3, 6, 12, 24, and 48 hours by injecting air from a calibration syringe into the circuit with all ports capped. We closed the exhalation valve by injecting 20 mL air into its supply line. A one-way valve was placed between the syringe and inspiratory limb of the breathing circuit to isolate the circuit during compliance measurements. Air was injected until pressure reached 100 cm H_2O as indicated on the ventilator manometer. The exhalation valve was opened and the released volume was measured with the calibration analyzer (Fig. 2). C_{BC} was calculated by dividing the released volume by 100 cm H_2O.

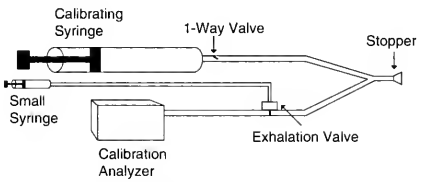

Fig. 2. Diagram showing method of measuring compressed volume using a calibrating syringe and calibration analyzer. The small syringe was used to inject air to close the exhalation valve of the circuit during compliance measurements.

We repeated the study, maintaining the circuits at room temperature in order to investigate the effects of time alone on C_{BC}.

Data were statistically analyzed using a two-way analysis of variance with time and circuit type as factors for both the heated and room-temperature circuit model and the level of significance set at 0.05. For the statistically significant analysis of variance, Tukey's honestly significant differences tests were performed to determine which means were significantly different from each other.[9,10]

Results

Under the conditions of warming described earlier, there was no significant change in the compliance of the EVA or the PBP circuits over 48 hours. However, the compliance of the HK circuit changed significantly during the first hour. These data are shown in Table 1. There was no significant difference in compliance of the HK circuit during the 2-48 hour time period ($p = 0.67$). At room temperature, there was no significant change in the compliance of

* Suppliers are identified in the Product Sources section at the end of the text.

Table 1. Mean (SD) Values for Circuit Compliance during Conditions of Heat and Room Temperature

	Heated			Unheated		
	At Setup	At 1 Hour	p Value	At Setup	At 48 Hours	p Value
Puritan-Bennett	1.47 (0.06)	1.47 (0.05)	0.24	1.46 (0.06)	1.47 (0.08)	0.24
Hudson EVA	1.50 (0.08)	1.51 (0.05)	0.26	1.51 (0.08)	1.52 (0.07)	0.26
Hudson kraton	2.31 (0.06)	2.52 (0.06)	< 0.001*	2.31 (0.07)	2.33 (0.06)	0.10

*Analysis of variance and Tukey's honestly significant differences test on the heated vs room-temperature circuit.

any of the circuits over 48 hours. These data are also shown in Table 1.

There was no significant difference in C_{BC} between the heated and unheated EVA ($p = 0.26$) or PBP ($p = 0.31$) circuits. The difference between the heated and unheated conditions of the HK circuit was significant ($p < 0.001$).

Discussion

This study was designed to determine whether breathing circuit compliance changed over time in three different types of disposable breathing circuits under conditions of heat and room temperature. The compliance values we measured were similar to those reported by Campbell et al[3] (Table 2), although we did not find any significant difference in C_{BC} of the heated EVA and PBP circuits during the first hour, which is while the circuits were being heated to 34°C. None of the previous studies[1,2,4,5] investigated the compliance of these particular BC brands. Cote and colleagues[5] studied only anesthesia-type circuits (Q and Mapelson) that are not commonly used in the intensive care unit. Forbat and Her[4] included the volume compressed within the ventilator making their estimates of C_{BC} much higher than ours. The range of values for C_{BC} reported by Hess et al[2] and Bartel et al[1] were 1.4-2.6 mL/cm H_2O and 0.9-3.8 mL/cm H_2O, respectively, and similar to those we report. The difference in BC brands and evaluation methods among our studies may account for the differences in results.

Table 2. Mean (SD) Breathing Circuit Compliance Values (mL/cm H_2O)—Our Data and Those of Campbell et al[3]

Circuit	Condition	Campbell et al	Valeri et al
Puritan-Bennett	Unheated	1.68 (0.1)	1.47 (0.06)
	Heated	1.96 (0.2	1.46 (0.06)
Hudson EVA	Unheated	1.78 (0.1)	1.50 (0.08)
	Heated	1.84 (0.2)	1.51 (0.08)
Hudson kraton	Unheated	3.24 (0.2)	2.31 (0.06)*
	Heated	3.54 (0.3)	2.52 (0.06)

*Analysis of variance and Tukey's honestly significant differences test on the heated vs room-temperature circuit.

With the HK circuits, we found a significant increase in compliance at the end of the first hour. This increase may be due to the heating of the circuit, as reported by Campbell et al,[3] and not to any changes that might have occurred due to some other mechanism (eg, wear from repeated stretching) over time. We found no significant changes in C_{BC} of unheated circuits over a 48-hour period. However, we saw a significant increase in C_{BC} of the HK circuit between the heated and unheated conditions at all time periods. These results support the conclusion that the difference in C_{BC} of the heated HK circuit was due to the heating of the circuit, and not to changes that occur over time. Although this difference in C_{BC} was statistically significant, it may not result in clinically important changes in delivered V_T. For example, at a set V_T of 0.8 L with a peak airway pressure of 40 cm H_2O, the volume lost to circuit compliance of the HK circuit would be 92 mL in the unheated circuit and 101 mL in the heated circuit, a difference of only 9 mL (1% of set V_T). However, with the use of small V_Ts, that 9 mL difference in compressed volume could be important (eg, 9 mL/180 mL = 5%). We also found that the breathing circuit compliance did not change significantly over a 48-hour period when using either the EVA or the PBP circuits. Therefore, it may not be necessary to recalculate breathing circuit compliance for the purpose of correcting V_T delivered to the patient for up to 48 hours of use. We found no change in C_{BC} of the HK circuit after the first hour. Considering the above caveats, when using HK, it may be prudent to recalculate the C_{BC} once the circuit has reached its operating temperature.

Additional investigations of the effect of humidity, airway pressure, and other stresses on compliance when applied to these and other circuits are indicated. Future researchers may find it enlightening to examine changes in C_{BC} when the circuits are humidified. Due to the limitations imposed by our test-lung system, we were unable to humidify the circuits we tested. One author has raised the question of whether breathing circuits may be used for extended periods (a week or more in certain circumstances),[11] and examination of circuit performance beyond the 48-hour time period used in our study may be useful. A

clinical study may also be useful in determining the effects of changes in breathing circuit compliance on the V_T actually delivered to the patient. Because we investigated only three BC brands, further study of other commercially available BCs is required to determine their specific characteristics.

Although we found a statistically significant increase in the compliance of one of the three circuits we tested (HK), there were no clinically important changes in the compliance of any of the breathing circuits over a 48-hour period. Therefore it may not be necessary to recalculate breathing circuit compliance for the purpose of correcting the set V_T to ensure delivered V_T.

ACKNOWLEDGMENTS

We thank Paula M Bentley RRT and Da'Henri R Thurmond RRT for their participation in data collection for this study.

PRODUCT SOURCES

Breathing Circuits:

Puritan-Bennett Polyethylene Volume Ventilator Circuit, No. 003011, Puritan-Bennett Corp, Lenexa KS

Hudson Ethylene Vinyl Acetate Universal Ventilator Circuit, No. 1627, Hudson Respiratory Care Inc, Temecula CA

Hudson Kraton Universal Volume Ventilator Circuit, No. 690-50, Hudson Respiratory Care Inc, Temecula CA

Ventilators:

MA2+2, Puritan-Bennett Corp, Carlsbad CA

BEAR 5, Bear Medical Systems, Riverside CA

7200 series, Puritan-Bennett Corp, Carlsbad CA

Lung Simulator:

Vent Aid TTL, Model 1600, Michigan Instruments Inc, Grand Rapids MI

Incubator:

Airshields Isolette Infant Incubator, Model C-86, Narco Health Co, Hatboro PA

Calibration Analyzer:

Timeter RT-200, Allied Healthcare Products Inc, St Louis MO

Calibration Syringe:

Hamilton Model S-1000, Hamilton Medical Inc, Reno NV

Statistical Software:

Execustat v.2.0, Strategy Plus Inc, available from PWS-Kent Publishing, Boston MA

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CORRECTION

In the April 1994 issue of the Journal [Respir Care 1994;39(4):347-362], an error was made in Table 4. The third equation (from Dillard et al, Reference 25) was incorrect. The correct equation should be as shown below.

$$0.453\ (P_{aO2ground}) + (0.386)\ (\%\ \text{predicted}\ FEV_1) + 2.440$$

We regret the error.

AARC Clinical Practice Guideline

Ventilator Circuit Changes

VCC 1.0 PROCEDURE:

Changing the mechanical ventilator circuit in settings other than the home, a component procedure of continuous mechanical ventilation

VCC 2.0 DESCRIPTION/DEFINITION:

The ventilator circuit change consists of placing a clean ventilator circuit on an operating mechanical ventilator. Ventilator circuits may be either disposable (designed for single-patient use) or reusable and may or may not contain heated wires. The ventilator circuit may consist of various components: gas delivery tubing (large bore), monitoring tubing (small bore), humidifier, heat-and-moisture exchanger (HME, which in this guideline includes hygroscopic condenser humidifiers, or HCHs), humidifier water reservoir, thermal monitoring probes, machine filters, spirometer, water traps, medication delivery device (small volume nebulizer, MDI chambers or adapters), large volume nebulizer, or 'secondary' humidifiers with gas reservoirs as components of continuous or parallel IMV gas delivery circuits.

2.1 The objectives of the ventilator circuit change procedure as described are

2.1.1 to limit the occurrence of nosocomial infection acquired as a consequence of endotracheal intubation or tracheostomy and ventilatory support;

2.1.2 to assure that the ventilator-circuit system maintains its physical integrity and proper function;

2.1.3 to provide a circuit that is clean in appearance;

2.1.4 to minimize the risk of harm to the patient and health care personnel during the process of changing the circuit.

VCC 3.0 SETTING:

This Guideline is confined to the institutional setting.

3.1 Intensive care units
3.2 Acute and extended care units
3.3 Skilled nursing facilities

VCC 4.0 INDICATIONS:

The decision to change a ventilator circuit should be governed by

4.1 length of time the existing circuit has been in use;[1-11]
4.2 type of circuit and humidification device in use;[1,3,6-20]
4.3 circuit function (presence of a malfunctioning circuit or a circuit that leaks);
4.4 appearance of ventilator circuit (circuits that are not clean in appearance should be replaced).

VCC 5.0 CONTRAINDICATIONS:

5.1 Presence of conditions in the patient's cardiopulmonary or neurologic status that might make tolerance of disconnection from mechanical ventilation hazardous to the patient
5.2 Inability to safely and effectively ventilate or maintain patient during the ventilator circuit change
5.3 Absence of a clean and functional circuit to use as a replacement

VCC 6.0 HAZARDS/COMPLICATIONS:

6.1 Patient's condition may predispose him or her to harm or injury during the changing process:

6.1.1 hemodynamic instability,

6.1.2 hypo- or hyperoxia with sequelae,

6.1.3 hyper- or hypocapnia,

6.1.4 airway obstruction,

6.1.5 artificial airway displacement,

6.1.6 contamination of patient or staff from exposure to material in circuit.[8,21-28]

6.2 Patient may not be safely maintained during disconnection from the ventilator:

6.2.1 inappropriate or inadequate ventilation (f and/or V_T),

6.2.2 inappropriate or inadequate oxygenation (F_{IO_2} and/or PEEP),

6.2.3 inappropriate increase in work of breathing,

6.2.4 airway obstruction.

6.3 Inability to assure that the replacement circuit has been safely and effectively disinfected and that it is operationally sound[29,30]

6.3.1 Transmission of pathogens to patient and to health care personnel

6.3.2 Hazards of exposure to residual toxic disinfectants or associated disinfectant by-products[30]

6.3.3 Malfunctioning or suboptimally functioning ventilator or circuit

6.3.4 Failure to assure proper ventilator function with patient reconnection (ie, correct settings, absence of leaks, functioning alarms, proper valve placement[31])

6.3.5 Potential for patient-ventilator disconnection[31]

6.4 Manipulation and disconnection of the ventilator tubing can cause contaminated ventilator condensate to spill into the patient's airway, exposing the patient to further risk of infection.[8,21-24,26-28]

6.5 Changing ventilator circuits more frequently than is necessary may increase the risk of nosocomial pneumonia.[8,23]

6.6 Failure to assure proper ventilator function prior to reinstituting mechanical ventilation may endanger patient.[31]

VCC 7.0 LIMITATIONS OF PROCEDURE:

7.1 Evidence suggests that routine circuit changes (more frequently than every 48 hours) do not provide any advantage in controlling infections acquired by patients while being mechanically ventilated.[3-5,13,23,28,32]

7.2 Controversy exists concerning the frequency at which ventilator circuits should be changed to minimize patient infection.[1-11,13-20,23,24,26,32-36]

7.3 Controversy exists concerning the influence that circuit design and the presence of various circuit components have on the safe interval of circuit change.

7.3.1 The type of humidification device used may influence circuit change interval.[8,12-15-20,24,26,33-37]

7.3.2 Use of heated vs non-heated circuits may influence circuit change interval.[6,7,9,10]

7.3.3 Use of disposable vs reusable circuits may influence circuit change interval.[4,24,26,27]

7.3.4 Use of aerosol generators (nebulizers) may influence circuit change interval.[2,21,29,37]

7.4 Changing ventilator circuits too frequently may incur added patient or institutional expense and deliver no additional infectious disease protection to the patient.[4,6,11-23]

VCC 8.0 ASSESSMENT OF NEED:

8.1 Objective—limiting the transmission of infection

8.1.1 Reliance on published Centers for Disease Control and Prevention (CDC) recommendations[25,29]

8.1.2 Reliance on institutional standards established by monitoring and surveillance and/or published research

8.2 Objective—to prevent malfunction and to maintain optimal performance

8.2.1 Competency of the circuit should be monitored for tubing leaks.

8.2.2 In-line filters should be assessed for increased resistance.[33-37]

8.2.3 Equipment affected over time by water (eg, spirometers) should be monitored.

8.3 Objective—to maintain a circuit clean in appearance: Inspect appearance of circuit.

CC 9.0 ASSESSMENT OF OUTCOME:

he following should be utilized to evaluate the enefit of the ventilator circuit change.

9.1 An ongoing infectious disease surveillance program, to include

9.1.1 Surveillance of incidence of nosocomial pneumonia in mechanically ventilated patients in the institution

9.1.2 Surveillance of institutional compliance with stated institutional policy on infection control in mechanically ventilated patients

9.2 An ongoing equipment-malfunction surveillance program

9.3 An operational verification, as described in the AARC Patient-Ventilator System Check CPG, immediately following circuit change to assure physical integrity and proper function of system[36]

VCC 10.0 RESOURCES:

10.1 Equipment

10.1.1 Changing the circuit

10.1.1.1 A clean (disinfected) and operational circuit including all of its various components[29,30]

10.1.1.2 A sterile source of water if required for humidification[29]

10.1.1.3 An appropriate device for maintenance of patient ventilation during circuit change (manual ventilation device or mechanical ventilator), including PEEP maintenance if indicated

10.1.2 Surveillance program

10.1.2.1 appropriate bacterial surveillance equipment and records

10.1.2.2 appropriate ventilator surveillance equipment and records

10.2 Personnel

10.2.1 Level II—trained personnel (preferably possessing the RRT or CRTT credential or a comparable credential) with demonstrated ability to apply, adjust, and troubleshoot the ventilator, provide alternative ventilation, and monitor the patient

10.2.2 Level II—trained personnel with knowledge of and demonstrated ability to apply techniques described in 10.2.1 and bacterial surveillance techniques

10.2.3 Quality assessment or quality improvement personnel/committee or physician—personnel qualified to interpret the significance of the results of bacterial surveillance

VCC 11.0 MONITORING:

Monitoring of the ventilator circuit change should also include observation of and appropriate response to

11.1 patient's hemodynamic values: blood pressure, heart rate, and rhythm;

11.2 patient's oxygenation: S_{pO_2}, and P_{aO_2} and S_{aO_2} (if available);

11.3 patient's acid-base and ventilatory status: P_{CO_2}, and pH and end-tidal CO_2 (if available);

11.4 patient's chest excursion, color, appearance, comfort;

11.5 stability of artificial airway and its placement;

11.6 complete patient-ventilator system check.[36]

VCC 12.0 FREQUENCY:

Controversy exists regarding the most desirable and effective interval for circuit change (One report found no difference in infection rates with 48-hour circuit changes versus no change[8]). The literature suggests that the particular type of equipment used may influence the desirable circuit change interval (see Sections 7.1 and 12.2). The 1981 CDC recommendation to change ventilator circuits every 24 hours was based largely on well-documented nosocomial infection of patients from *Pseudomonas* species in the humidifier reservoir and was established before innovations such as heat moisture exchangers, newer non-aerosolizing humidifiers, and heated wire circuits were in wide use. Research suggests that the patient infects the ventilator circuit, and the ability for the circuit to grow bacteria and infect the patient is limited by potential routes of transmission (eg, aerosols and contaminated condensate).[28] The most important routes of transmission are via improper hand-washing techniques and

gastrointestinal tract colonization.[28,29]

12.1 It is recommended that circuit change intervals be established based on published standards, evaluation of published clinical research literature, community standards of practice, and/or a well-designed local surveilance program. If a closed-system suction setup is included, it should not be used longer than the circuit and should be replaced as necessary when found to be nonfunctional or soiled in appearance.

12.2 Guideline recommendations are based on the equipment used:

12.2.1 Circuits employing nebulizers (aerosol generators) as humidifiers require 24-hour change interval.[2,3,29,37]

12.2.2 Careful evaluation of the literature suggests that circuits employing heated Cascade-type devices ('bursting-bubble' humidifiers) as humidifiers,[3,5,12,13,16-20,22,26,37] circuits employing heat moisture exchangers (HME), or artificial noses, and circuits employing wick or other vapor-phase humidifiers may be changed at an interval ≥ 5 days.[6,10,12,15,26,33] HMEs should be changed every 24 hours or more often if necessary.[14-20,38]

12.2.2.1 Change HME as required to provide proper function.

12.2.2.2 Change to maintain a circuit clean in appearance.

VCC 13.0 INFECTION CONTROL:

As discussed elsewhere in this Guideline, control of the infectious risk of the ventilator to the patient is a foremost objective of the ventilator circuit change.

13.1 Personnel should observe Universal Precautions when involved in circuit changes,[25,29] including the use of gloves, protective eyewear, and liquid-barrier gowns.

13.2 Liquid contained within the circuit is contaminated and should be handled properly, disposed of as medical waste (ie, contaminated), and should not be drained into the humidification device.[21,22,24,26,27,29,32]

13.3 Circuit humidifiers should be filled with sterile water.[29]

13.3.1 Aseptic technique should be followed when filling these devices.[29]

13.3.2 Closed circuit filling devices or continuous-feed systems for humidifiers or nebulizers may be desirable because they limit interruptions to the circuit.[23]

Mechanical Ventilation Guidelines Committee:

Robert S Campbell RRT, Chairman, Tampa FL
Richard D Branson RRT, Cincinnati OH
William "Chuck" Burke PhD RRT, Indianapolis IN
Jack Covington RRT, San Francisco CA

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American Association for Respiratory Care
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AARC Clinical Practice Guideline

Delivery of Aerosols to the Upper Airway

AUA 1.0 PROCEDURE:

Administration of aerosols to the upper airway

AUA 2.0 DESCRIPTION:

Delivery of therapeutic aerosols to the upper airway including the nose, pharynx, and larynx—therapeutic aerosols are indicated for upper airway inflammation, anesthesia, rhinitis, and administration of medications for systemic effect.

Devices for upper airway delivery include metered dose inhalers (MDI), metered spray pumps (MSP), hand-bulb atomizers (HBA), and small-volume (SVN), large-volume (LVN), and ultrasonic nebulizers (USN).

Nasopharyngeal deposition is greatest for particles in the range of 5-20 μ.[1]

AUA 3.0 SETTING:

Bland or pharmacologically active aerosol therapy can be administered in a number of settings, including hospital, clinic, diagnostic laboratory, extended care facility, and home.

AUA 4.0 INDICATIONS:

4.1 Upper airway inflammation (eg, to relieve inflammation due to laryngotracheobronchitis[2])
4.2 Anesthesia (eg, to control pain and gagging during endoscopic procedures[3-7])
4.3 Rhinitis (eg, to relieve inflammation and vascular congestion[8,9])
4.4 Systemic disease (eg, to deliver peptides such as insulin[10])

AUA 5.0 CONTRAINDICATIONS:

Known hypersensitivity to the medication being delivered

AUA 6.0 HAZARDS/COMPLICATIONS:

6.1 Administration of medications for upper airway inflammation may result in
- **6.1.1** bronchospasm,[11,12]
- **6.1.2** rebound of symptoms,
- **6.1.3** systemic side effects.

6.2 Administration of medications for anesthesia[3-7] may result in
- **6.2.1** inhibition of gag reflex,
- **6.2.2** choking,
- **6.2.3** dehydration of epithelium,
- **6.2.4** allergic reactions,
- **6.2.5** excessive systemic effect,
- **6.2.6** bronchospasm,
- **6.2.7** nosocomial infection from contaminated delivery device or medication (See AARC CPG on Selection of Aerosol Delivery Device[13]).[14]

6.3 Administration of medications for rhinitis[15-21] may result in
- **6.3.1** nasal rebound (including rhinitis medicamentosa) after extended use of alpha adrenergic decongestants;
- **6.3.2** delayed effect (eg, effects of steroids are not immediate);
- **6.3.3** sensation of irritation and burning in the nose;
- **6.3.4** sneezing attacks (immediately following administration);
- **6.3.5** mucosal ulceration and bleeding;
- **6.3.6** postnasal drip.

6.4 Systemic disease—medication may cause nasal irritation or toxic effects.[9,10]

AUA 7.0 LIMITATIONS OF PROCEDURE:

7.1 Upper airway inflammation—patient cooperation is not essential but may be desirable for effective administration in some applications.
7.2 Anesthesia—direct local application of anesthetic agents may require direct visualization.
7.3 Rhinitis
7.3.1 In the presence of excessive nasal mucus secretion or edema of the nasal mucosa, the drug may fail to reach the site of intended action. Deposition may be compromised by the presence of nasal polyps or a deviated septum.
7.3.2 Response may not be readily observable for drugs such as cromolyn sodium and steroids.
7.4 Systemic disease
7.4.1 In the presence of excessive nasal mucus secretion or edema of the nasal mucosa, the drug may fail to reach the site of intended action. Deposition may also be compromised by the presence of nasal polyps or a deviated septum.
7.4.2 Response may not be immediately apparent.

AUA 8.0 ASSESSMENT OF NEED:

The presence of one or more of the following may suggest the need for aerosol application.

8.1 In upper airway inflammation—
8.1.1 stridor
8.1.2 brassy croup-like cough
8.1.3 hoarseness following extubation
8.1.4. diagnosis of laryngeotracheobronchitis or croup
8.1.5 recent extubation
8.1.6 evidence of inflamed upper airway
8.1.7 soft-tissue radiograph suggesting edema
8.1.8 increased work of breathing
8.2 For anesthesia—
8.2.1 severe localized pain in upper airway
8.2.2 impending invasive instrumentation of the upper airway
8.3 In rhinitis—
8.3.1 diagnosis of allergic, non-allergic, or infectious rhinitis
8.3.2 symptomatic need:
8.3.2.1 nasal congestion
8.3.2.2 rhinorrhea
8.3.2.3 sneezing
8.3.2.4 itching of nose, eyes, or palate
8.4 In systemic disease—presence of a systemic disease that warrants intranasal delivery of a therapeutic agent

AUA 9.0 ASSESSMENT OF OUTCOME:

9.1 In upper airway inflammation, effectiveness of administration may be indicated by
9.1.1 reduced stridor,
9.1.2 reduced hoarseness,
9.1.3 improvement in soft-tissue radiograph,
9.1.4 decreased work of breathing, as evidenced by decreased use of accessory muscles.
9.2 For anesthesia, effectiveness of administration is marked by reduced discomfort in the patient.
9.3 In rhinitis, effectiveness of administration may be indicated by
9.3.1 reduced nasal congestion,
9.3.2 improved airflow through nose,
9.3.3 reduced rhinorrhea,
9.3.4 reduced sneezing,
9.3.5 reduced itching of nose, eyes, or palate.
9.4 Systemic disease—effectiveness of administration of agent is marked by the presence of the appropriate systemic therapeutic response.

AUA 10.0 RESOURCES:

10.1 Equipment:
10.1.1 metered dose inhaler, or MDI, with nasal adapter
10.1.2 metered spray pump, or MSP
10.1.3 hand-bulb atomizer, or HBA
10.1.4 small volume nebulizer, or SVN, (use requires gas source, flowmeter, connecting tubing, and mask or mouthpiece)
10.1.5 large volume nebulizer, or LVN, (use requires gas source, flowmeter, large-

bore tubing, and mask or mouthpiece)

10.1.6 ultrasonic nebulizer, or USN, (use requires electrical source, large-bore tubing and mouthpiece, mask, or nasal adapter)

10.2 Personnel:

10.2.1 A Level I caregiver may be the provider of service after Level II personnel have established need for a specific device by patient assessment, initial administration(s) has been completed, and patient is considered stable. Level I personnel (typically CRTT or RRT) must demonstrate appropriate technique for

10.2.1.1 preparing and measuring medications;

10.2.1.2 administering medication;

10.2.1.3 equipment use;

10.2.1.4 cleaning equipment;

10.2.1.5 disposal of wastes;

10.2.1.6 encouraging effective breathing patterns;

10.2.1.7 modifying treatment in response to adverse reactions as instructed, with appropriate communication with physician and Level II caregiver, in response to severity of symptoms;

10.2.1.8 Universal Precautions and proper infection control

10.2.2 Level II personnel should perform initial assessments of all patients and care for the unstable patient. The Level II caregiver should be an RRT or have equivalent training in patient assessment, aerosol administration, pulmonary function, and lung mechanics and should have demonstrated his/her knowledge and ability to perform related to the following:

10.2.2.1 indications and limitations for MDI, MSP, MBA, SVN, LVN, and USN;

10.2.2.2 proper use, maintenance, and cleaning of equipment, including filter and scavenging systems;

10.2.2.3 risks inherent in the medications and specific devices;

10.2.2.4 procedures for safely disposing of medical wastes;

10.2.2.5 breathing patterns and coughing techniques;

10.2.2.6 technique modification in response to adverse reactions;

10.2.2.7 dose and/or frequency modification in response to severity of symptoms, as prescribed;

10.2.2.8 assessment of patient condition and response to therapy;

10.2.2.9 auscultation, inspection, and vital-signs determination;

10.2.2.10 measurement of peak expiratory flowrate, flow-volumes, timed volumes, and ventilatory mechanics;

10.2.2.11 recognition and response to adverse reactions to and complications of medication and/or procedure;

10.2.2.12 instructing patients and families.

AUA 11.0 MONITORING:

The extent of patient monitoring should be determined on the basis of the stability and severity of the patient's condition:

11.1 patient compliance and increase or decrease in symptoms, signs, and patient subjective response as specified in Section 8;

11.2 heart rate and rhythm, blood pressure;

11.3 change in indicators of therapeutic effect (eg, blood-glucose level with insulin).

AUA 12.0 FREQUENCY:

Frequency of administration is specific for purpose of administration and medication used.

AUA 13.0 INFECTION CONTROL:

13.1 Universal Precautions for body fluid isolation are to be implemented.[22]

13.2 Centers for Disease Control and Prevention recommendations for control of exposure to tuberculosis and droplet nuclei are to be implemented when patient is known to have tuberculosis or has other risk factors for the disease.[23]

13.2.1 To reduce aerosol contamination of room air,

13.2.1.1 enclose and contain aerosol ad-

ministration;

13.2.1.2 filter aerosols that bypass or are exhaled by patient;

13.2.2 When aerosol release to the atmosphere cannot be routed through a filter,

13.2.2.1 use filtered scavenger systems to remove aerosols that cannot be contained;

13.2.2.2 provide local exhaust ventilation to remove aerosols that are released into room air;

13.2.2.3 provide frequent air exchange to dilute concentration of aerosol in room;

13.2.2.4 allow exchange of gas in the room to eliminate 99% of aerosol before next patient enters or receives treatment in that area.

13.2.3 It remains unclear whether high efficiency particulate air (HEPA) filters completely remove infectious particles from the air.

13.2.4 Filters, nebulizers, and other contaminated disposable components of the aerosol delivery system should be treated as medical waste. Applicator nozzles for multidose atomizers should be changed between patients.

13.2.5 Personal protection devices should be used to reduce exposure when engineering alternatives are not adequate or in place.[23]

13.2.5.1 Use properly fitted respirator with adequate filtration when flow exhaust cannot control removal of aerosols.

13.2.5.2 Goggles, gloves, masks, and gowns should be used to serve as splatter shields and to reduce exposure to body substances.

13.2.6 Personnel should safely dispose of hazardous wastes.

13.2.7 Personnel who are at high risk for adverse effects from exposure should be offered an opportunity for alternative assignments.

13.3 Nebulizers should not be reused between patients without disinfection.

13.4 Nebulizers should be changed or sterilized

13.4.1 at conclusion of a procedure that is not to be repeated;

13.4.2 every 24 hours with repeated or continuous administration or more often when visibly soiled;

13.5 Nebulizers ***should not*** be rinsed with tap water between treatments.[24,25]

13.6 Solutions should be handled aseptically.

13.7 Diluent solutions from multidose vials (eg, distilled water and normal saline) must be handled aseptically and should be discarded after 24 hours.

Aerosol Therapy Guidelines Committee:

Jon Nilsestuen PhD RRT, Chairman, Houston TX
James B Fink MBA RRT, Chicago IL
James K Stoller MD, Cleveland OH
James Volpe RRT, San Diego CA
Theodore Witek Jr DrPH RPFT RRT, Ridgefield CT

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AARC Clinical Practice Guideline

Neonatal Time-Triggered, Pressure-Limited, Time-Cycled Mechanical Ventilation

TPTV 1.0 PROCEDURE:

The application of time-triggered, pressure-limited, time-cycled mechanical ventilation (TPTV) to neonates—this Guideline does not address patient-triggered ventilation.

TPTV2.0 DESCRIPTION/DEFINITION:

The application of time-triggered, pressure-limited, time-cycled mechanical ventilation in the neonate is typically accomplished by the use of commercially available pressure-limited ventilators specifically designed for this population or of multipurpose, multimodal ventilators with the necessary capabilities. These ventilators permit precise management of ventilator settings.[1-8]

Pressure-limited ventilators commonly incorporate continuous flow to deliver a mixture of oxygen and air. The continuous flow supplies the patient with a fresh gas source for spontaneous breathing.[7,9-14]

Mandatory breaths are time-triggered and time-cycled, based on an adjustable frequency and inspiratory time. As the ventilator time-triggers inspiration, the exhalation valve closes, and the continuous flow is directed to the inspiratory limb of the patient circuit for the length of the inspiratory time.[7,11,12,14]

Once the pressure limit has been reached, remaining flow is diverted to a limiting mechanism. As the ventilator cycles to expiration, the exhalation valve opens to ambient and the continuous flow exits the expiratory limb of the circuit.[7,10,14]

Flow continues to enter the patient's lungs until pressure equilibrates or until the inspiratory time has elapsed, resulting in a decelerating flow pattern. Volume delivery is dependent on lung and chest-wall compliance and airway resistance, including the resistance of the endotracheal tube. As these variables change, deceleration of flow is altered, and the tidal volume (V_T) varies.[10,11,15-22] The V_T achieved depends on the pressure limit, gas flowrate, inspiratory time (t_I), and positive end-expiratory pressure (PEEP). Circuit characteristics that affect V_T include compressible volume, presence of condensate, and obstruction of the artificial airway.[3,4,8-10,18,20,23]

TPTV 3.0 SETTINGS:

TPTV is applied by trained personnel in acute care and subacute care hospitals.

TPTV 4.0 INDICATIONS:

The presence of one or more of the following conditions constitutes an indication for TPTV.

4.1 Apnea [24-27]

4.2 Respiratory or ventilatory failure, despite the use of continuous positive airway pressure (CPAP) and supplemental oxygen (ie, $F_{IO_2} \geq 0.60$)[24,25,28]

- **4.2.1** Respiratory acidosis with a pH < 7.20-7.25[8,25,29]
- **4.2.2** $P_{aO_2} < 50$ torr[8,13,25,29,30]
- **4.2.3** Abnormalities on physical examination
  - **4.2.3.1** Increased work of breathing demonstrated by grunting, nasal flaring, tachypnea, and sternal and intercostal retractions[5,27,29,31]
  - **4.2.3.2** The presence of pale or cyanotic skin and agitation

4.3 Alterations in neurologic status that compromise the central drive to breathe:

- **4.3.1** Apnea of prematurity[32]
- **4.3.2** Intracranial hemorrhage[33]

4.3.3 Congenital neuromuscular disorders[34]

4.4 Impaired respiratory function resulting in a compromised functional residual capacity (FRC) due to decreased lung compliance and/or increased airways resistance,[12,35] including but not limited to

4.4.1 Respiratory distress syndrome (RDS)[1,2,28,36-39]

4.4.2 Meconium aspiration syndrome (MAS)[40]

4.4.3 Pneumonia[41]

4.4.4 Bronchopulmonary dysplasia[42-44]

4.4.5 Bronchiolitis[41]

4.4.6 Congenital diaphragmatic hernia[45]

4.4.7 Sepsis[41]

4.4.8 Radiographic evidence of decreased lung volume[27]

4.5 Impaired cardiovascular function

4.5.1 Persistent pulmonary hypertension of the newborn (PPHN)[46,47]

4.5.2 Postresuscitation[41]

4.5.3 Congenital heart disease[48]

4.5.4 Shock[6]

4.6 Postoperative state characterized by impaired ventilatory function[45,49]

TPTV 5.0 CONTRAINDICATIONS:

No specific contraindications for neonatal TPTV exist when indications are judged to be present (Section 4.0).

TPTV 6.0 HAZARDS/COMPLICATIONS:

6.1 Air leak syndromes due to barotrauma and/or volume overinflation (ie, volutrauma),[2,11,50-54] including

6.1.1 Pneumothorax[16,55-59]

6.1.2 Pneumomediastinum[55,56,58]

6.1.3 Pneumopericardium[55]

6.1.4 Pneumoperitoneum[55]

6.1.5 Subcutaneous emphysema[55]

6.1.6 Pulmonary interstitial emphysema[56,60-62]

6.2 Chronic lung disease associated with prolonged positive pressure ventilation and oxygen toxicity[63,64] (eg, bronchopulmonary dysplasia[42,43,65-68])

6.3 Airway complications associated with endotracheal intubation

6.3.1 Laryngotracheobronchomalacia[69]

6.3.2 Damage to upper airway structures[66,69,70]

6.3.3 Malpositioning of endotracheal tube (ETT)[69,71]

6.3.4 Partial or total obstruction of ETT with mucus[69,71,72]

6.3.5 Kinking of ETT[69,71]

6.3.6 Unplanned extubation[69,71,73]

6.3.7 Air leak around uncuffed ETT

6.3.8 Subglottic stenosis[69]

6.3.9 Main-stem intubation[69]

6.3.10 Pressure necrosis[74]

6.3.11 Increased work of breathing (during spontaneous breaths) due to the high resistance of endotracheal tubes of small internal diameter[75]

6.4 Nosocomial pulmonary infection (eg, pneumonia[76])

6.5 Complications that occur when positive pressure applied to the lungs is transmitted to the cardiovascular system[77,78] or the cerebral vasculature resulting in

6.5.1 Decreased venous return[77,78]

6.5.2 Decreased cardiac output[77,78]

6.5.3 Increased intracranial pressure leading to intraventricular hemorrhage[27,33,79]

6.6 Supplemental oxygen in conjunction with TPTV may lead to an increased risk of retinopathy of prematurity (ROP)[80,81]

6.7 Complications associated with endotracheal suctioning[82]

6.8 Technical complications

6.8.1 Ventilator failure[10,83]

6.8.2 Ventilator circuit and/or humidifier failure[38,70] (Condensate in the inspiratory limb of the ventilator circuit may result in a reduction in V_T[23,84] or inadvertent pulmonary lavage.)

6.8.3 Ventilator alarm failure[10,38,69,83]

6.8.4 Loss of or inadequate gas supply

6.9 Patient-ventilator asynchrony[85,86]

6.10 Inappropriate ventilator settings leading to

6.10.1 auto-PEEP

6.10.2 hypo-or hyperventilation

6.10.3 hypo- or hyperoxemia

6.10.4 increased work of breathing

TPTV 7.0 LIMITATIONS OF METHOD:

Rapid changes in lung compliance and airways resistance may result in alterations in V_T delivery that may significantly alter minute ventilation ($\dot{V}_E$).[3,4,9,11,19-21]

TPTV 8.0 ASSESSMENT OF NEED:

Determination that valid indications are present by physical, radiographic, and laboratory assessment

TPTV 9.0 ASSESSMENT OF OUTCOME:

Establishment of neonatal assisted ventilation should result in improvement in patient condition and/or reversal of indications (Section 4.0):

9.1 Reduction in work of breathing as evidenced by decreases in respiratory rate, severity of retractions, nasal flaring and grunting

9.2 Radiographic evidence of improved lung volume[41]

9.3 Subjective improvement in lung volume as indicated by increased chest excursion and aeration by chest auscultation[87]

9.4 Improved gas exchange

9.4.1 Ability to maintain a $P_{aO_2} \geq 50$ torr with $F_{IO_2} < 0.60$[8,30]

9.4.2 Ability to reverse respiratory acidosis and maintain a pH > 7.25[8,30]

9.4.3 Subjective improvement as indicated by a decrease in grunting, nasal flaring, sternal and intercostal retraction, and respiratory rate[31]

TPTV 10.0 RESOURCES:

10.1 Equipment—recommended equipment based on individual patient need includes the following. (For a given patient, all mentioned equipment may not be necessary.)

10.1.1 Commercially available continuous-flow infant ventilator equipped with TPTV mode or suitably equipped multipurpose, multimodal ventilator, low and high airway pressure alarms, high-pressure release-to-ambient pressure capability, loss of power and gas source alarms, low and high oxygen concentration alarms—alarms may be integral to the ventilator or an add-on.[21,38,83]

10.1.2 Servo-regulated humidifier with low compressible volume chamber and preferably a continuous water source[21,38,83]

10.1.3 Low-compliance infant ventilator circuit with heated inspiratory and expiratory wires compatible with the servo-regulated humidification system is recommended.[19,23,38,83,88,89] Comparable circuits that permit continual drainage of condensate may also be used.

10.1.4 Endotracheal tube with associated intubation equipment or tracheostomy tube with associated insertion and cleaning accessories and supplies for securing the tube

10.1.5 Suction source, suction catheters, and normal saline for instillation to ensure patency of artificial airway[82]

10.1.6 Standby resuscitation apparatus with airway manometer and masks of appropriate size[83] and supplies for chest-tube insertion

10.1.7 Continuous noninvasive monitoring of oxygenation by either transcutaneous monitor or pulse oximeter with high- and low-alarm capabilities[90,91]

10.1.8 Continuous noninvasive monitoring of carbon dioxide by transcutaneous CO_2 monitor with high- and low-alarm capabilities is also recommended[38,92] for the unstable baby requiring frequent ventilator changes. Measurements of end-tidal CO_2 may also be useful in these infants.[93,94]

10.1.9 Continuous cardiorespiratory monitoring (eg, ECG and respiratory rate) with high- and low-alarm capabilities[38]

10.1.10 Appropriate and adequate compressed gas supply

10.1.11 Graphic display of airway pressure, flow, and tidal volume may be useful.

10.2 Personnel: TPTV should be applied under the direction of a physician by trained personnel who hold a recognized credential (eg, CRTT, RRT, RN) and who competently demonstrate

10.2.1. proper use, understanding, and mastery of the technical aspects of management of artificial airways, mechanical

ventilators, and humidification systems;

10.2.2 comprehensive knowledge of ventilator management and understanding of neonatal airway anatomy and pulmonary pathophysiology;

10.2.3 patient assessment skills, with an understanding of the interaction between the mechanical ventilator and the patient and ability to recognize and respond to adverse reactions and complications;

10.2.4 knowledge and understanding of intubation equipment;

10.2.5 ability to interpret monitored and measured blood gas parameters and vital signs;

10.2.6 application of Universal Precautions;[92]

10.2.7 proper use, understanding, and mastery of emergency resuscitation equipment and procedures;

10.2.8 ability to evaluate and document results of outcome assessments (Section 9.0);

10.2.9 ability to interpret chest radiographs to determine proper placement of artificial airways and identify complications associated with mechanical ventilation (eg, air leak syndromes).

TPTV 11.0 MONITORING:

11.1 Patient-ventilator system checks should be performed every 2-4 hours and should include documentation of ventilator settings and patient assessments as recommended by the AARC CPG Patient-Ventilator System Checks (MV-SC) and AARC CPG Humidification during Mechanical Ventilation (HMV).[95,96]

11.2 Oxygen and CO_2 monitoring

11.2.1 Periodic sampling of blood gas values by arterial, capillary, or venous route.[1,24,30] P_{aO_2} should be kept below 80 torr in preterm infants to minimize the risk of ROP.[6,80,97]

11.2.2 The unstable infant should be monitored continuously by transcutaneous O_2 monitor or pulse oximeter.[1,90,91]

11.2.3 The unstable infant should be monitored continuously by transcutaneous[90] or end-tidal CO_2 monitoring.[93,94]

11.2.4 Fractional concentration of oxygen delivered by the ventilator should be monitored continuously.[38]

11.3 Continuous monitoring of cardiac activity (via electrocardiograph) and respiratory rate[98]

11.4 Monitoring of blood pressure by indwelling arterial line or by periodic cuff measurements[24]

11.5 Continuous monitoring of proximal airway pressures including peak inspiratory pressure (PIP), PEEP, and mean airway pressure ($\overline{P}_{aw}$)[1,39,99]

11.5.1 Increases in $\overline{P}_{aw}$ may result in improved oxygenation; however, $\overline{P}_{aw} > 12$ cm H_2O has been associated with barotrauma.[8,20,41,99-102]

11.5.2 The difference between PIP and PEEP (ΔP) in conjunction with patient mechanics determines V_T. As the ΔP changes, V_T will vary.[16,51,99,100]

11.5.3 PIP should be adjusted initially to achieve adequate V_T as reflected by chest excursion and adequate breath sounds[8,29,100] and/or by V_T measurement.

11.5.4 PEEP increases FRC and may improve oxygenation and ventilation-perfusion relationship (PEEP is typically adjusted at 4-7 cm H_2O—levels beyond this range may result in hyperinflation, particularly in patients with obstructive airways disease [eg, MAS or bronchiolitis][5,29,35,103-105]).

11.6 Many commercially available neonatal ventilators provide continuous monitoring of ventilator frequency, t_I, and I:E. If only two of these variables are directly monitored, the third should be calculated (eg, the proportion of the t_I for a given frequency determines the I:E).

11.6.1 Lengthening t_I increases $\overline{P}_{aw}$ and should improve oxygenation.[1,2,13,24,41,106,107]

11.6.2 I:E in excess of 1:1 may lead to the development of auto-PEEP and hyperinflation.[5,20,24,37,105,106,108]

11.6.3 Frequencies of 30-60 per minute with shorter t_I (eg, I:E of 1:2) are commonly used in patients with RDS.[8,22,41,50,59,85,109-111]

11.7 Depending on the internal diameter of the ventilator circuit, excessive flowrates can result in expiratory resistance that leads to increased

work of breathing and automatic increases in PEEP.[17,19,89,98,86,112] Some ventilators are equipped with demand-flow systems that permit the use of lower baseline flowrates but provide the patient with additional flow as needed

11.8 Because of the possibility of complete obstruction or kinking of the ETT and the inadequacy of ventilator alarms in these situations, continuous tidal volume monitoring via an appropriately designed (minimum dead space) proximal airway flow sensor is recommended.[98,113,114]

11.9 Periodic physical assessment of chest excursion and breath sounds and for signs of increased work of breathing and cyanosis.[3,5,21,87]

11.10 Periodic evaluation of chest radiographs to follow the progress of the disease, identify possible complications, and verify ETT placement[21,27,79]

TPTV 12.0 FREQUENCY:

TPTV is intended for continuous use and is discontinued when the patient's clinical condition improves as indicated by results of outcome assessments (Section 9.0).

TPTV 13.0 INFECTION CONTROL:

No special precautions are necessary, but Universal Precautions as described by the Centers for Disease Control should be employed.[95]

13.1 Ventilator circuits and humidifier chambers should not be changed more frequently than every 48 hours. The Clinical Practice Guideline: Ventilator Circuit Changes, the CDC, and, reported experience[114-116] suggest that use periods of ≤ 5 days are acceptable when the humidifying device is other than an aerosol generator.

13.2 External surfaces of ventilator should be cleaned according to the manufacturer's recommendations when the device has remained in a patient's room for a prolonged period, when soiled, when it has come in contact with potentially transmittable organisms, and after each patient use.

13.3 Sterile suctioning procedures should be strictly adhered to.[82]

Perinatal-Pediatrics Guidelines Committee:

Lynne K Bower RRT, Chairman, Boston MA
Sherry L Barnhart RRT, Mattoon IL
Peter Betit BS RRT, Boston MA
Barbara Hendon BA RRT RCP, Wylie TX
Joanne Masi-Lynch BS RRT, Salt Lake City UT
Barbara G Wilson MEd RRT, Durham NC

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AARC Clinical Practice Guideline

Application of Continuous Positive Airway Pressure to Neonates via Nasal Prongs or Nasopharyngeal Tube

NCPAP 1.0 PROCEDURE:

The application of continuous positive airway pressure to neonates and infants by nasal prongs (NCPAP) or by nasopharyngeal tube (NP-CPAP) used in conjunction with a commercially available continuous-flow infant ventilator or a suitably equipped multipurpose ventilator

NCPAP 2.0 DESCRIPTION/DEFINITION:

Continuous positive airway pressure (CPAP) is the application of positive pressure to the airways of the spontaneously breathing patient throughout the respiratory cycle.[1-4] For the most part, neonates are nose breathers; therefore, the application of nasal CPAP is easily facilitated.[5,6] This is accomplished by inserting nasopharyngeal tubes or affixing nasal prongs to the patient. The device is attached to a warmed and humidified gas from a continuous-flow mechanical ventilator designed for neonates or a suitably equipped multipurpose ventilator, set in the CPAP mode.[7-13] Freestanding systems are sometimes used; however, their use is not addressed in this guideline.

CPAP maintains inspiratory and expiratory pressures above ambient pressure,which should result in an increase in functional residual capacity (FRC) and improvement in static lung compliance and decrease airway resistance in the infant with unstable lung mechanics.[1,3,14-20] This allows a greater volume change/unit of pressure change (ie, greater tidal volume for a given pressure change) with subsequent reduction in the work of breathing and stabilization of minute ventilation ($\dot{V}_E$).[9,16,18,19,21-23] CPAP increases mean airway pressure, and the associated increase in FRC should improve ventilation-perfusion relationships and potentially reduce oxygen requirements.[15,16,18,19,21,24-28]

NCPAP 3.0 SETTINGS:

NCPAP or NP-CPAP is applied by trained personnel in acute and subacute care hospitals.

NCPAP 4.0 INDICATIONS:

4.1 Abnormalities on physical examination—the presence of increased work of breathing as indicated by a 30-40% increase above normal in respiratory rate, substernal and suprasternal retractions, grunting, and nasal flaring;[16,27,29] the presence of pale or cyanotic skin color and agitation[16]

4.2 Inadequate arterial blood gas values—the inability to maintain a P_{aO_2} greater than 50 torr with F_{IO_2} of ≤ 0.60 provided $\dot{V}_E$ is adequate as indicated by a P_{aCO_2} level of 50 torr and a pH ≥ 7.25[9,11,14]

4.3 The presence of poorly expanded and/or infiltrated lung fields[16,19] on chest radiograph

4.4 The presence of a condition thought to be responsive to CPAP and associated with one or more of the clinical presentations in 4.1-4.3[18]

4.4.1 Respiratory distress syndrome[9,11,14]

4.4.2 Pulmonary edema[30]

4.4.3 Atelectasis[16]

4.4.4 Apnea of prematurity[27,31-34]

4.4.5 Recent extubation[35,36]

4.4.6 Tracheal malacia or other similar abnormality of the lower airways[9,37,38]

4.4.7 Transient tachypnea of the newborn[16]

NCPAP 5.0 CONTRAINDICATIONS:

5.1 Although NCPAP and NP-CPAP have been used in bronchiolitis,[39] this application may be contraindicated.[40]

5.2 The need for intubation and/or mechanical ventilation as evidenced by the presence of

5.2.1 Upper airway abnormalities that

make NCPAP or NP-CPAP ineffective or potentially dangerous (eg, choanal atresia, cleft palate, tracheoesophageal fistula)[41]

5.2.2 Severe cardiovascular instability and impending arrest

5.2.3 Unstable respiratory drive with frequent apneic episodes resulting in desaturation and/or bradycardia

5.2.4 Ventilatory failure as indicated by the inability to maintain $P_{aCO_2} < 60$ torr and pH > 7.25[25]

5.3. Application of NCPAP or NP-CPAP to patients with untreated congenital diaphragmatic hernia may lead to gastric distention and further compromise of thoracic organs.[41]

NCPAP 6.0 HAZARDS/COMPLICATIONS:

6.1 Hazards and complications associated with equipment include the following.

6.1.1 Obstruction of nasal prongs from mucus plugging or kinking of nasopharyngeal tube may interfere with delivery of CPAP and result in a decrease in F_{IO_2} through entrainment of room air via opposite naris or mouth.

6.1.2 Inactivation of airway pressure alarms

6.1.2.1 Increased resistance created by turbulent flow through the small orifices of nasal prongs and nasopharyngeal tubes can maintain pressure in the CPAP system even when decannulation has occurred. This can result in failure of low airway pressure/disconnect alarms to respond.[5]

6.1.2.2 Complete obstruction of nasal prongs and nasopharyngeal tubes results in continued pressurization of the CPAP system without activation of low or high airway pressure alarms.

6.1.2.3 Activation of a manual breath (commonly available on infant ventilators) may cause gastric insufflation and patient discomfort particularly if the peak pressure is set inappropriately high.[31]

6.2 Hazards and complications associated with the patient's clinical condition include

6.2.1 lung overdistention leading to

6.2.1.1 air leak syndromes,[42,43]

6.2.1.2 ventilation-perfusion mismatch,[15]

6.2.1.3 CO_2 retention and increased work of breathing,[5,44]

6.2.1.4 impedance of pulmonary blood flow with a subsequent increase in pulmonary vascular resistance and decrease in cardiac output.[30,45]

6.2.2 gastric insufflation and abdominal distention potentially leading to aspiration.[46]

6.2.3 nasal irritation with septal distortion.

6.2.4 skin irritation and pressure necrosis.

6.2.5 nasal mucosal damage due to inadequate humidification.

NCPAP 7.0 LIMITATIONS OF DEVICE:

7.1 NCPAP and NP-CPAP applications are not benign procedures, and operators should be aware of the possible hazards and complications and take all necessary precautions to ensure safe and effective application.

7.2 NCPAP and NP-CPAP are ineffective during mouth breathing, resulting in loss of desired pressure and decrease in delivered oxygen concentration.[10,47,48]

7.3 NCPAP harnesses and attachment devices are often cumbersome and difficult to secure and may cause agitation and result in inadvertent decannulation.[5,47]

NCPAP 8.0 ASSESSMENT OF NEED:

Determination that valid indications are present by physical, radiographic, and laboratory assessments

NCPAP 9.0 ASSESSMENT OF OUTCOME:

CPAP is initiated at levels of 4-5 cm H_2O and may be gradually increased up to 10 cm H_2O to provide the following:[9,17,27,28,47-50]

9.1 Stabilization of F_{IO_2} requirement ≤ 0.60 with P_{aO_2} levels > 50 torr and/or the presence of clinically acceptable noninvasive monitoring of oxy-

gen (P_{tcO_2}),[7,25,48,49,51] while maintaining an adequate $\dot{V}_E$ as indicated by P_{aCO_2} of 50-60 torr or less and pH ≥ 7.25.

9.2 Reduction in the work of breathing as indicated by a decrease in respiratory rate by 30-40% and a decrease in the severity of retractions, grunting, and nasal flaring.[16,27,29]

9.3 Improvement in lung volumes and appearance of lung as indicated by chest radiograph.[27]

9.4 Improvement in patient comfort as assessed by bedside caregiver.

NCPAP 10.0 RESOURCES:

10.1 Equipment

10.1.1 Endotracheal tubes (positioned in the nasopharynx and secured by taping, with placement verified by laryngoscopy or palpation) or commercially available nasal prongs or nasopharyngeal tubes with accompanying harness and accessories may be used for CPAP administration.

10.1.2 Commercially available continuous-flow infant ventilators equipped with CPAP mode or suitably equipped multipurpose ventilator, intregral or adjunct low and high airway pressure alarms, low and high oxygen concentration alarms, loss of power and gas source alarms may be used.[52]

10.1.3 Lightweight ventilator circuits with servo-regulated humidification system

10.1.4 Continuous noninvasive oxygenation monitoring by pulse oximetry or transcutaneous monitor with high and low alarm capabilities is recommended (continuous transcutaneous CO_2 monitoring may also be utilized).[53,54]

10.1.5 Continuous electrocardiographic and respiratory rate monitor, with high and low alarm capabilities, is recommended.

10.1.6 Suction source, suction regulator, and suction catheters for periodic suctioning to assure patency of nasal passages and of endotracheal tubes used for NP-CPAP are necessary.[55]

10.1.7 Resuscitation apparatus with airway manometer and masks of appropriate size should be available.

10.1.8 Gastric tube for periodic decompression of stomach and chest tubes should be available.

10.2 Personnel: The application of NCPAP and NP-CPAP should be performed under the direction of a physician by trained personnel who hold a recognized credential (eg, CRTT, RRT, RN) and who competently demonstrate

10.2.1 proper use, understanding, and mastery of the technical aspects of CPAP devices, mechanical ventilators, and humidification systems;

10.2.2 knowledge of ventilator management and understanding of neonatal airway anatomy and pulmonary physiology;

10.2.3 patient assessment skills, with an understanding of the interaction between the CPAP device and the patient and the ability to recognize and respond to adverse reactions and complications;

10.2.4 knowledge and understanding of artificial airway management, training in the procedures of placing endotracheal tubes in the nasopharynx;

10.2.5 the ability to interpret monitored and measured blood gas values and vital signs;

10.2.6 the application of Universal Precautions;[56]

10.2.7 proper use, understanding, and mastery of emergency resuscitation equipment and procedures;

10.2.8 the ability to assess, evaluate, and document outcome (Section 9.0).

NCPAP 11.0 MONITORING:

11.1 Patient-ventilator system checks should be performed at least every 2 to 4 hours and should include documentation of ventilator settings and patient assessments as recommended by the AARC CPG Patient-Ventilator System Checks (MV-SC) and the CPG Humidification during Mechanical Ventilation (HMV).[57,58]

11.2 Oxygen and carbon dioxide monitoring, including

11.2.1 Periodic sampling of blood gas values by arterial, capillary, or venous

route[7,9,14,27,28,59,60]

11.2.2 Continuous noninvasive blood gas monitoring by transcutaneous O_2 and CO_2 monitors[27,60]

11.2.3 Continuous noninvasive monitoring of oxygen saturation by pulse oximetry[27,38]

11.3 Continuous monitoring of electrocardiogram and respiratory rate[24,25,27]

11.4 Continuous monitoring of proximal airway pressure (P_{aw}), PEEP, and mean airway pressure ($\bar{P}_{aw}$)[24,25,27]

11.5 Continuous monitoring of F_{IO_2}[24,25,27]

11.6 Periodic physical assessment of breath sounds and signs of increased work of breathing (see Section 4.1)[16,24,25]

11.7 Periodic evaluation of chest radiographs[24,27,56]

NCPAP 12.0 FREQUENCY:

NCPAP and NP-CPAP are intended for continuous use and are discontinued when the patient's clinical condition improves as indicated by successful outcome assessments (Section 9.0).

NCPAP 13.0 INFECTION CONTROL:

No special precautions are necessary, but Universal Precautions[56] as described by the Centers for Disease Control should be employed.

13.1 Disposable nasal CPAP kits are recommended and are intended for single-patient use.

13.2 Ventilator circuits and humidifier chambers should not be changed more frequently than every 48 hours. The Clinical Practice Guideline: Ventilator Circuit Changes, the CDC, and, reported experience[61-64] suggest that use periods of $\leq$ 5 days are acceptable when the humidifying device is other than an aerosol generator.

13.3 External surfaces of ventilator should be cleaned according to the manufacturer's recommendations when the device has remained in a patient's room for a prolonged period, when soiled, when it has come in contact with potentially transmittable organisms, and after each patient use.

13.4 Sterile suctioning procedures should be strictly adhered to.[5,55]

Perinatal-Pediatrics Guidelines Committee:

Lynne K Bower RRT, Chairman, Boston MA
Sherry L Barnhart RRT, Mattoon IL
Peter Betit BS RRT, Boston MA
Jamie Clink RRT, Dallas TX
Barbara Hendon BA RCP RRT, Wylie TX
Joanne Masi-Lynch BS RRT, Salt Lake City UT
Barbara G Wilson MEd RRT, Durham NC

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AARC Clinical Practice Guideline

Surfactant Replacement Therapy

SRT 1.0 PROCEDURE:

Surfactant replacement therapy in the neonate

SRT 2.0 DESCRIPTION/DEFINITION:

Natural, endogenous surfactant is a compound composed of phospholipids, neutral lipids, and proteins[1-5] that forms a layer between the alveolar surface and the alveolar gas and reduces alveolar collapse by decreasing surface tension within the alveoli.[3-5] Surfactant deficiency is almost always associated with the formation of hyaline membranes in the immature lung and the onset of respiratory distress syndrome (RDS)—a major cause of morbidity and mortality in premature infants.[3] Without surfactant, alveoli may never inflate or may collapse on expiration and require inordinate force to re-expand on inspiration, leading to the development of RDS.[3,5] The incidence of RDS is related more to lung immaturity than to gestational age.[6] However, in general, the more premature the infant, the less the surfactant production and the higher the probability for RDS.[4,6] Direct tracheal instillation of surfactant has been shown to reduce mortality and morbidity in infants with RDS.[7-25]

Surfactant can be extracted from animal lung lavage and from human amniotic fluid or produced from synthetic materials.

Two basic strategies for surfactant replacement have emerged: (1) prophylactic or preventive treatment in which surfactant is administered at the time of birth or shortly thereafter to infants who are at high risk for developing RDS and (2) rescue or therapeutic treatment in which surfactant is administered after the initiation of mechanical ventilation in infants with clinically confirmed RDS.[2,10-12,26,27]

SRT 3.0 SETTINGS:

Administered by trained personnel in

3.1 Delivery room
3.2 Neonatal intensive care unit

SRT 4.0 INDICATIONS:

4.1. Prophylactic administration may be indicated in

4.1.1 infants at high risk of developing RDS because of short gestation (< 32 weeks)[8,10-12,21,25-29] or low birthweight (< 1,300 g),[21-25,28] which strongly suggest lung immaturity.

4.1.2 infants in whom there is laboratory evidence of surfactant deficiency such as lecithin/sphingomyelin ratio less than 2:1,[11,14,28,30,31] bubble stability test indicating lung immaturity,[15,32] or the absence of phosphatidylglycerol.[11,14,22-24,28,30]

4.2 Rescue or therapeutic administration is indicated in preterm or full-term infants

4.2.1 who require endotracheal intubation and mechanical ventilation because of

4.2.1.1 increased work of breathing as indicated by an increase in respiratory rate, substernal and suprasternal retractions, grunting, and nasal flaring.[8,11,14-16,29,33-35]

4.2.1.2 increasing oxygen requirements as indicated by pale or cyanotic skin color, agitation, and decreases in P_{aO_2}, S_{aO_2}, or S_{pO_2} mandating an increase in F_{IO_2} above 0.40[11,12,15,26,33,36-38]

and

4.2.2 have clinical evidence of RDS,[13,39] including

4.2.2.1 chest radiograph characteristic of RDS,[8,11-16,33,34,36,37,40-42]

4.2.2.2 mean airway pressure greater than 7 cm H_2O to maintain an adequate P_{aO_2}, S_{aO_2}, or S_{pO_2}.[11,14,15,26,43]

SRT 5.0 CONTRAINDICATIONS:

Relative contraindications to surfactant administration are

5.1 the presence of congenital anomalies incompatible with life beyond the neonatal period,[8,14,15,26,28,29,31,33,36,41,44]

5.2 respiratory distress in infants with laboratory evidence of lung maturity.[9,14,27-29,33,36,41]

SRT 6.0 HAZARDS/COMPLICATIONS:

6.1 Procedural complications resulting from the administration of surfactant include

6.1.1 plugging of endotracheal tube (ETT) by surfactant;[2]

6.1.2 hemoglobin desaturation and increased need for supplemental O_2;[11,33,41]

6.1.3 bradycardia due to hypoxia;[9,33,41,45]

6.1.4 tachycardia due to agitation, with reflux of surfactant into the ETT;[34,41]

6.1.5 pharyngeal deposition of surfactant;

6.1.6 administration of surfactant to only one lung;

6.1.7 administration of suboptimal dose secondary to miscalculation or error in reconstitution.

6.2 Physiologic complications of surfactant replacement therapy include

6.2.1 apnea,[7,13,15]

6.2.2 pulmonary hemorrhage,[12,15,18,32,34,38,46,47]

6.2.3 mucus plugs,[48]

6.2.4 increased necessity for treatment for PDA,[18,29,30]

6.2.5 marginal increase in retinopathy of prematurity,[11]

6.2.6 barotrauma resulting from increase in lung compliance following surfactant replacement and failure to change ventilator settings accordingly.[30,49]

SRT 7.0 LIMITATION OF METHOD:

7.1 Surfactant administered prophylactically may be given to some infants in whom RDS would not have developed.[10,12,26,33]

7.2 When surfactant is administered prophylactically in the delivery room, ETT placement may not have been verified by chest radiograph resulting in the inadvertent administration to only one lung or to the stomach.[26]

7.3 Prophylactic surfactant administration may delay patient stabilization.[26]

7.4 Atelectasis and lung injury may occur prior to therapeutic administration.[26,33]

7.5 Tracheal suctioning should be avoided following surfactant administration.[9,11,13,14,27,33,38,44,50]

7.6 Not all infants who are treated with a single dose of surfactant experience a positive response[39] or the response may be transient.

7.7 Positioning recommended for surfactant administration may further compromise the unstable infant.[9,11,12,14,16,28,33,38-40]

SRT 8.0 ASSESSMENT OF NEED:

Determine that valid indications are present.

8.1 Assess lung immaturity prior to prophylactic administration of surfactant by gestational age and birthweight and/or by laboratory evaluation of tracheal or gastric aspirate.

8.2 Establish the diagnosis of RDS by chest radiographic criteria and the requirement for mechanical ventilation in the presence of short gestation and/or low birthweight.

SRT 9.0 ASSESSMENT OF OUTCOME:

9.1 Reduction in F_{IO_2} requirement[12,33,34,36-39,41,44]

9.2 Reduction in work of breathing[51]

9.3 Improvement in lung volumes and lung fields as indicated by chest radiograph[13,16,33,40]

9.4 Improvement in pulmonary mechanics (eg, compliance, airways resistance, V_T, $\dot{V}_E$, transpulmonary pressure) and lung volume (ie, FRC)[42,43,50,52-59]

9.5 Reduction in ventilator requirements (PIP, PEEP, $\overline{P}_{aw}$)[2,8,9,12,13,27,30,33,36-39,41,44,50,52]

9.6 Improvement in ratio of arterial to alveolar P_{O_2} (a/A P_{O_2}), oxygen index[13,16,28,30,33,34,37-41,44]

SRT 10.0 RESOURCES:

Administration procedures recommended for specific preparations of surfactant should be adhered to.

10.1 Equipment[10-14,16,26-28,33,34,39,40,50,60]

10.1.1 Administration equipment

10.1.1.1 Syringe containing the ordered dose of surfactant, warmed to room temperature[11,12,16,38,40]

10.1.1.2 5-Fr feeding tube or catheter, or endotracheal tube connector with delivery port

10.1.1.3 Mechanical ventilator or manual ventilator (resuscitation bag)[8,16,33,36,38-40,44,50,52]

10.1.2 Resuscitation equipment

10.1.2.1 Laryngoscope and endotracheal tube[10-12,14,16,26,38]

10.1.2.2 Manual resuscitation bag[9-12,16,26-28,36,39,40,50] and airway manometer

10.1.2.3 Blended oxygen source[9,16,28,44]

10.1.2.4 Suction equipment (ie, catheters, sterile gloves, collecting bottle and tubing, and vacuum generator)[9,33,50,60]

10.1.2.5 Radiant warmer ready for use

10.1.3 Monitoring equipment

10.1.3.1 Neonatal tidal volume monitor if available[50]

10.1.3.2 Airway pressure monitor

10.1.3.3 Pulse oximeter or transcutaneous P_{CO_2} monitor[11,26,34,39-41,52]

10.1.3.4 Cardiorespiratory monitor

10.2 Personnel—Surfactant replacement therapy should be performed under the direction of a physician by credentialed personnel (eg, CRTT, RRT, RN) who competently demonstrate

10.2.1 proper use, understanding, and mastery of the equipment and technical aspects of surfactant replacement therapy;

10.2.2 comprehensive knowledge and understanding of neonatal ventilator management and pulmonary anatomy and pathophysiology;

10.2.3 neonatal patient assessment skills, including the ability to recognize and respond to adverse reactions and/or complications of the procedure;

10.2.4 knowledge and understanding of the patient's history and clinical condition;

10.2.5 knowledge and understanding of airway management;

10.2.6 ability to interpret monitored and measured blood gas variables and vital signs;

10.2.7 proper use, understanding, and mastery of emergency resuscitation equipment and procedures;

10.2.8 ability to evaluate and document outcome (Section 9.0);

10.2.9 understanding and proper application of Universal Precautions.

SRT 11.0 MONITORING:

The following should be monitored as part of surfactant replacement therapy.

11.1 Variables to be monitored during surfactant administration

11.1.1 Proper placement and position of delivery device

11.1.2 F_{IO_2} and ventilator settings[8,9,11,13-15,27-29,33,36,38,44]

11.1.3 Reflux of surfactant into ETT[34,41]

11.1.4 position of patient (ie, head direction)[9,11,33]

11.1.5 Chest-wall movement[61]

11.1.6 Oxygen saturation by pulse oximetry[11,26,34,39-41,52]

11.1.7 Heart rate, respirations, chest expansion, skin color, and vigor[16,26,27,34,41,45,52]

11.2 Variables to be monitored after surfactant administration

11.2.1 Invasive and noninvasive measurements of arterial blood gases[8,9,11,12-16,26-29,33,36,38,39,41,44]

11.2.2 Chest radiograph[11-16,28,36,38-40,44]

11.2.3 Ventilator settings (PIP, PEEP, $\overline{P}_{aw}$) and F_{IO_2}[8,9,11,13-16,28,29,33,36,38]

11.2.4 Pulmonary mechanics and volumes

11.2.5 Heart rate, respirations, chest expansion, skin color, and vigor[16,26,27,34,41,45,52]

11.2.6 Breath sounds[11,38]

11.2.7 Blood pressure[13,16,33,40,44,45]

SRT 12.0 FREQUENCY:

Repeat doses of surfactant are contingent upon the continued diagnosis of RDS. The frequency with which surfactant replacement is performed should depend upon the clinical status of the patient and the indication for performing the procedure. Additional doses of surfactant, given at 6- to 24-hour intervals, may be indicated in infants who experience increasing ventilator requirements or whose conditions fail to improve after the initial dose.[7,9,11,12,14,15,26,30,34,37,39,52]

SRT 13.0 INFECTION CONTROL:

13.1 Universal Precautions[62] should be implemented.
13.2 Aseptic technique should be practiced.
13.3 Appropriate infection control guidelines for the patient should be posted and followed.

Perinatal-Pediatrics Guidelines Committee:

Lynne K Bower RRT, Chairman, Boston MA
Sherry L Barnhart RRT, Mattoon IL
Peter Betit BS RRT, Boston MA
Barbara Hendon BA RRT RCP, Wylie TX
Joanne Masi-Lynch BS RRT, Salt Lake City UT
Barbara G Wilson MEd RRT, Durham NC

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AARC Clinical Practice Guideline

Static Lung Volumes

SLV 1.0 PROCEDURE:

Measurement of static lung volumes and capacities in children (age ≥ 5) and adults—this guideline focuses on commonly used techniques for measuring lung volumes, including spirometry, gas-dilution determination of functional residual capacity (FRC), and whole-body plethysmography determination of thoracic gas volume (TGV). Other methods (eg, single-breath nitrogen, single-breath helium, and roentgenologic determinations of lung volumes) are not discussed.

SLV 2.0 DESCRIPTION/DEFINITIONS:

2.1 Static lung volumes are determined using methods in which airflow velocity does not play a role. The sum of two or more lung-volume subdivisions comprises a lung capacity. The subdivisions and capacities are expressed in liters at body temperature and pressure saturated with water vapor (BTPS).

2.2 Tidal volume (V_T, TV—both terms have been used interchangeably. TV is used in this guideline) is the volume of air that is inhaled or exhaled with each respiratory cycle.[1] It varies with the conditions (eg, rest, exercise, posture). An average of at least 6 breaths should be used.[2]

2.3 Inspiratory reserve volume (IRV) is the maximal volume of air that can be inhaled from the tidal-volume end-inspiratory level.[2]

2.4 Expiratory reserve volume (ERV) is the maximal volume of air that can be exhaled after a normal tidal exhalation (ie, from functional residual capacity, or FRC).[2]

2.5 Residual volume (RV) is the volume of gas remaining in the lung at the end of a maximal expiration.[1] It is usually calculated by subtracting the ERV from FRC (RV = FRC – ERV), but can also be obtained by subtracting vital capacity (VC) from total lung capacity, or TLC (RV = TLC – VC).[2]

2.6 Inspiratory capacity (IC) is the maximal volume of air that can be inhaled from the tidal-volume end-expiratory level (ie, FRC). It is equal to the sum of TV and IRV.[2]

2.7 Vital capacity (VC) is the volume change that occurs between maximal inspiration and maximal expiration. The subdivisions of the VC include tidal volume (TV), inspiratory reserve volume (IRV), and expiratory reserve volume (ERV). The largest of three technically satisfactory vital capacities should be reported. The two largest vital capacities should agree within 5% or 100 mL, whichever is larger.[2,3] The volume change can be accomplished in several ways:[2]

2.7.1 Inspiratory vital capacity (IVC)—a slow maximal inspiration after a slow maximal expiration;

2.7.2 Expiratory vital capacity (EVC):—a slow maximal expiration after a slow maximal inspiration;

2.7.3 Two-stage vital capacity:—a slow maximal inspiration from tidal-volume end-expiratory level after a normal exhaled tidal volume, followed by quiet breathing, followed by a slow maximal expiration from tidal volume (ie, end-expiratory level, or functional residual capacity, or FRC)—the reverse is also acceptable;

2.7.4 Forced vital capacity (FVC)—the volume of air exhaled during a forced maximal expiration following a forced maximal inspiration.

2.8 FRC is the volume of air in the lung at the average tidal-volume end-expiratory level. It is the sum of the expiratory reserve and residual volumes. The method of measurement should be specified (eg, helium dilution, nitrogen washout, body plethysmography).[2]

2.9 Thoracic gas volume (TGV, V_{TG}) is the volume of air in the thorax at any point in time and at any level of thoracic compression. It is usually measured by whole-body plethysmography. It may be determined at any level of lung inflation; however, it is most commonly determined at FRC.[2]

2.10 Total lung capacity (TLC) is the volume of air in the lung at the end of a maximal inspiration. It can be calculated in two ways: (1) TLC = RV + VC, or (2) TLC = FRC + IC. The method of measurement (eg, gas dilution, body plethysmographyy) should be specified.[2]

SLV 3.0 SETTINGS:

The measurement of static lung volumes is performed in the pulmonary function laboratories of hospitals, clinics, and physicians' offices. Additionally, static lung volume determinations may be performed in patient care areas.

SLV 4.0 INDICATIONS:

Indications include but are not limited to the need

4.1 to differentiate between obstructive and restrictive disease patterns;[2]

4.2 to assess response to therapeutic interventions[2] (eg, drugs, transplantation, radiation, chemotherapy, lobectomy);

4.3 to aid in the interpretation of other lung function tests;[2]

4.4 to make preoperative assessments[2] in patients with compromised lung function (known or suspected) when the surgical procedure is known to affect lung function;

4.5 to evaluate pulmonary disability;[2]

4.6 to quantify the amount of nonventilated lung.

SLV 5.0 CONTRAINDICATIONS:

5.1 No apparent absolute contraindications exist; the relative contraindications for spirometry are appropriate and include the following:[4]

5.1.1 hemoptysis of unknown origin,

5.1.2 untreated pneumothorax,

5.1.3 unstable cardiovascular status,

5.1.4 thoracic and abdominal or cerebral aneurysms.

5.2 With respect to whole-body plethysmography, such factors as claustrophobia, upper body paralysis, obtrusive body casts, or other conditions that immobilize or prevent the patient from fitting into or gaining access to the 'body box' are a concern. In addition, the procedure may necessitate stopping intravenous therapy or supplemental oxygen.

SLV 6.0 HAZARDS/COMPLICATIONS:

6.1 Nosocomial infection contracted from improperly cleaned tubing, mouthpieces, and pneumotachographs

6.2 Discomfort associated with the body box

6.3 Depressed ventilatory drive in susceptible subjects (ie, CO_2 retainers) as a consequence of breathing 100% oxygen during the nitrogen washout,[5] such patients should be carefully observed

6.4 Hypercapnia and hypoxemia during helium-dilution FRC determinations as a consequence of failure to adequately remove CO_2 or add O_2

SLV 7.0 LIMITATIONS OF METHODOLOGY/VALIDATION OF RESULTS:

7.1 Patient-related limitations:

7.1.1 Spirometry is effort-dependent and requires understanding and motivation on the subject's part. Physical and/or mental impairment may limit patient's ability to perform.

7.1.2 Some patients cannot perform the necessary panting maneuver required for plethysmographic determination of FRC.

7.1.3 Some subjects are unable to maintain mouth seal or cooperate adequately for time necessary to perform the test.

7.1.4 Certain pathologic conditions in the subject can cause a leak in a lung volume measurement system (eg, perforated eardrum).

7.1.5 FRC measured by gas dilution may be underestimated in individuals with airflow limitation and air trapping.[6,7] It has also been shown that body plethysmogra-

phy can overestimate FRC in subjects with severe airway obstruction or induced bronchospasm.[8-10]

7.1.6 The elimination of nitrogen from tissues and body fluid can result in overestimation of the FRC in healthy subjects unless appropriate corrections are made.[2]

7.2 Test validation encompasses those calibration and procedural elements that help assure credible results.

7.2.1 Spirometry

7.2.1.1 Spirometers (volume-displacement devices or flow-sensing devices) should meet the American and European Thoracic Societies' current (1993) accepted standards.[2,3] Volume-displacement spirometers should be leak tested when calibrated (eg, daily).[11]

7.2.1.2 The VC (preferably an IVC) should be measured as close in time as possible to the FRC determination.[2]

7.2.2 Gas-dilution methods for FRC determination

7.2.2.1 Open circuit multibreath nitrogen washout method

7.2.2.1.1 Test should be continued for 7 minutes or until N_2 concentration falls below 1.0%.[12] In subjects with airflow obstruction and air trapping, the time period for measuring FRC should be prolonged.

7.2.1.1.2 A minimum of 15 minutes should elapse before test is repeated.[13]

7.2.1.1.3 Initial alveolar nitrogen concentration of 80% can be assumed,[2] if patient has been breathing room air for ≥ 15 minutes.

7.2.2.2 Closed circuit multibreath helium equilibration method

7.2.2.2.1 The helium concentration should be measured at least every 15 seconds, and water vapor should be removed from the fraction of gas that is introduced into the helium analyzer.[2]

7.2.2.2.2 A mixing fan should circulate and completely mix the air throughout the main circuit.

7.2.2.2.3 The breathing valve and mouthpiece (without a filter) should add only a small dead space to the system (< 60 mL for adult) and should easily disassemble for cleaning.

7.2.2.2.4 Gas mixing is considered complete when the change in helium concentration has been constant over a 2-minute period (ie, changes less than 0.02%) or 10 minutes has elapsed. If the helium concentration can be read directly or processed by computer, helium equilibration can be assumed when the change is < 0.02% in 30 seconds.[2]

7.2.2.2.5 The need for corrections for body absorption of helium is controversial.

7.2.2.2.6 At least 5 minutes should elapse before the test is repeated.

7.2.4 Whole body plethysmography

7.2.4.1 Panting breathing movements against the shutter should be made at a rate of 1 cycle/second.[8-10,14]

7.2.4.2 The cheeks and chin should be firmly supported with both hands.

7.2.4.3 Plethysmographic determination of FRC is the method of choice in patients with airflow limitation and air trapping.[2]

7.2.4.4 This method may be more practical in subjects with short attention spans or inability to stay on the mouthpiece (eg, children).

7.3 Reproducibility of results is essential to validation and test quality.

7.3.1 Multiple FRC determinations by gas dilution should be made, with at least two trials agreeing within 10%.[15]

7.3.2 FRC determinations by body plethysmography (at least 3 separate trials) should agree within 5%.[16]

7.3.3 The two largest IC and ERV measurements should agree within 5% or 60 mL whichever is larger.

7.3.4 The two largest VC measurements should agree within 5% or 100 mL whichever is larger.[3]

7.4 Clear and complete reporting of results is essential to test quality.

7.4.1 The average FRC value should always be reported (ideally including the variability).

7.4.2 The largest VC should be reported.

7.4.3 Either largest or mean values may be reported for IC and ERV.

7.4.4 Acceptable methods for reporting TLC and RV are

TLC = mean FRC + largest IC.
TLC = RV + VC.
RV = mean FRC – mean ERV.
RV = mean FRC – largest ERV.
RV = TLC – VC.

7.5 Conditions under which testing is done can affect results and should be held constant if possible. If conditions cannot be met, the written report should reflect that.

7.5.1 Lung volumes are influenced by body position[17,18] and should be made in the sitting position. If another position is used, it should be noted.[2]

7.5.2 Breathing movements should not be restricted by clothing.[2]

7.5.3 Diurnal variations in lung function may cause differences and, thus, if serial measurements will be performed, the time of the day that measurements are made should be held constant.[2]

7.5.4 The patient should not have smoked for at least 1 hour prior to the measurements.

7.5.5 The patient should not have had a large meal shortly before testing.

7.5.6 Nose clips should always be worn.[2]

7.5.7 Measurements made at ambient temperature and pressure saturated with water vapor (ATPS) conditions are corrected to body temperature and pressure saturated with water vapor (BTPS) conditions.

7.5.8 No corrections are necessary for altitude because no consistent differences in lung volumes (TLC, VC, FRC, and RV) due solely to altitude have been found from sea level up to 1,800 meters.[19-21]

7.5.9 The patient should be placed on the mouthpiece and asked to breathe quietly for 30 to 60 seconds, in order to become accustomed to the apparatus and attain a stable breathing pattern (ie, end-expiratory level reproducible within 100 mL).

7.5.10 The ERV, IC, and IVC can be measured before disconnecting the patient from measuring systems. Alternatively, the patient can be disconnected and the ERV, IC, and IVC performed immediately afterward.

7.5.11 If expired VC is measured with CO_2 absorber in the system, an appropriate volume correction should be made (ie, 1.05 × expired volume).

7.5.12 If a filter is used during FRC measurement, the filter volume must be subtracted.

7.5.13 When possible, lung volume measurements should precede forced expiratory maneuvers, which could induce bronchospasm and thus shift FRC and/or reduce VC.

7.6 Choice and application of reference values affect interpretation.

7.6.1 Make a tentative selection from published reference values. The characteristics of the healthy reference population should match the study group with respect to age, body size, gender, and racial and ethnic mix. The equipment, techniques, and measurement conditions should be similar.

7.6.2 Following selection of apparently appropriate reference values, compare measurements obtained from a representative sample of healthy individuals (10-20 subjects) over an appropriate age range to the predicted values obtained from the selected reference values. If an appreciable number of the sample falls outside of the normal range, more appropriate reference values should be sought. This procedure detects only relatively gross differences between sample and reference population.[22]

7.6.3 Predicted values for RV, FRC, and TLC should be derived from the same reference population.

7.7 Expression of results

7.7.1 The common practice of expressing

results as percent predicted and regarding 80% predicted as the lower limit of normal is not valid if the scatter of reference study data is independent of the level of lung function.[23]

7.7.2 Another method for describing the upper and lower limits of normal comes from the standard error of the estimates (SEE) around the regression lines. The 2-tail 95% confidence interval can be used and is determined by multiplying ± 1.96 times the SEE. The 2-tail 90% confidence interval can also be used and is determined by multiplying ± 1.64 times the SEE.[22,24]

SLV 8.0 ASSESSMENT OF NEED:

Determination that valid indications are present

SLV 9.0 ASSESSMENT OF OUTCOME/ TEST QUALITY:

Outcome and test quality are determined by ascertaining that the desired information has been generated for the specific indication and that validity and reproducibility have been assured.

9.1 Results are valid if the equipment functions acceptably and the subject is able to perform the maneuvers in an acceptable and reproducible fashion.

9.2 Report of test results should contain a statement, by the technician performing the test, about test quality (including patient understanding of directions and effort expended) and, if appropriate, which recommendations were not met.[2,3,25]

SLV 10.0 RESOURCES:

10.1 Equipment: Specifications should conform to recognized standards.

10.1.1 Spirometer specifications[2]

10.1.2 Pneumotachograph specifications[25]

10.1.3 Helium analyzer (katharometer) specifications[25]

10.1.4 Plethysmograph specifications[2]

10.1.5 Nitrogen analyzer specifications—range 0-100% ± 0.5% with 50 millisecond response time

10.2 Personnel

10.2.1 Lung volume testing should be performed under the direction of a physician specifically trained in pulmonary diagnostics.[26]

10.2.2 Personnel should be specifically trained (with verifiable training and demonstrated competency) in all aspects of lung volume determination, including equipment theory of operation, quality control, and test outcomes relative to diagnosis and/or medical history.[26]

10.2.3 At least one of the following credentials is recommended: CPFT, RPFT, CRTT, RRT.

SLV 11.0 MONITORING:

The following should be monitored during lung-volume determinations:

11.1 test data of repeated efforts (ie, reproducibility of results) to ascertain the validity of results.

11.2 the patient for any adverse effects of testing. (Patients on supplemental oxygen may require periods of time to rest on oxygen between trials.)

SLV 12.0 FREQUENCY:

The frequency of lung volume measurements depends on the clinical status of the subject and the indications for performing the test.

SLV 13.0 INFECTION CONTROL:

The measurement of lung volumes is a relatively safe procedure but a possibility of cross-contamination exists, from the patient-patient or patient-technologist interface.

13.1 Because of lack of evidence, the use of filters is neither recommended nor discouraged.[27]

13.1.1 If filters are used in gas-dilution procedures, their volume should be subtracted when FRC is calculated.

13.1.2 If filters are used in the plethysmo-

graph system, the resistance of the filters should be subtracted from the airways resistance calculation.

13.2 Nondisposable mouthpieces and equipment parts that come into contact with mucous membranes, saliva, and expirate should be cleaned and sterilized or subjected to high-level disinfection between patients.[28,29]

13.3 Flow sensors, valves, and tubing not in direct contact with the patient should be routinely disinfected according to the hospital's infection control policy.

13.4 Water-sealed spirometers should be drained weekly and allowed to dry.[2]

13.5 Centers for Disease Prevention and Control guidelines for control of tuberculosis should be followed.[30]

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Update on Inhaled Steroids

Hugh S Mathewson MD and Anthony L Kovac MD

Adrenocorticosteroids are regarded as indispensable agents for the prevention and treatment of asthma. This belief has been reinforced in recent years by the recognition of asthma as a chronic inflammatory disease.[1,2] Suppression of inflammation has become a primary goal for patients with bronchial hyperreactivity.[3,4] Within their therapeutic limitations, corticosteroids have assumed a central therapeutic role despite their manifold effects on metabolic processes.[5,6]

The untoward actions of corticosteroids are twofold—those resulting from continued use of large doses and those resulting from withdrawal. Prolonged therapy can induce fluid and electrolyte disturbances, hypertension, hyperglycemia, increased susceptibility to infections, peptic ulcers, osteoporosis, myopathy, cataracts, arrest of growth and healing processes, behavioral disturbances, and development of the characteristic habitus known as Cushing's syndrome.[7] Continued administration results in suppression of hypothalamic-pituitary-adrenal (HPA) function that can be slow in returning to normal when treatment is stopped. Too rapid withdrawal may bring on a condition characterized by fever, myalgia, arthralgia, and malaise, or in severe cases the shock-like syndrome of acute adrenal insufficiency.[7] The potency of the steroid compound and the degree of systemic uptake determine the extent to which metabolic changes occur.

The targets of therapeutic agents in asthma are the bronchial mucosal and smooth muscle cells. These can be reached by topically applied corticosteroids delivered by aerosol. If significant systemic absorption of drug can be avoided, the metabolic effects can be minimized.[8,9] Alternatively, these effects will be minor if the drug is rapidly inactivated upon absorption into the circulation. Both approaches have been utilized in constructing corticosteroid derivatives that are topically effective but which are either poorly absorbed or are promptly biodegraded by the liver. A similar rationale is applicable to the design of corticosteroids for application to the skin in the treatment of dermatoses.

To assess the topical effectiveness of corticosteroids, a skin test on human volunteers has been devised.[10] When applied to the skin, lipophilic steroids produce vasoconstriction. A known range of doses of the test steroid is applied to the skin of the inner forearm, and the blanching due to vasoconstriction is assessed visually in comparison with the laboratory standard, fluocinolone acetonide. The systemic activity of the compound may be measured in mice by noting the reduction in weight of the thymus gland 7 days after subcutaneous administration of the compound in oil.[11] The ratio of the vasoconstriction in man to thymolytic activity in the mouse can be used as an index of selectivity for topical use.

Inhaled corticosteroids can produce untoward local side effects. Oral candidiasis occurs in about one third of patients,[12] and is related to the frequency of administration and total dose of drug. Gargling with water after treatment or the use of a spacer may help to minimize yeast infection.[13] Nystatin, an antifungal agent, may be used in the gargle solution if indicated. Another side effect is dysphonia, which may affect up to one half of patients.[14] In some subjects, deposition of the corticosteroid on the vocal cords appears to cause bilateral adductor-muscle paresis from local myopathy, resulting in hoarseness.[15] This is also dose related.

Corticosteroids that are currently available for aerosol administration to asthmatic patients include beclomethasone dipropionate, triamcinolone acetonide, betamethasone valerate, flunisolide, budesonide, and fluticasone propionate. Dexamethasone sodium phosphate is also marketed, but its prolonged use has resulted in systemic side effects, including adrenal suppression.[16] Structural formulas of these compounds are shown in Figure 1. The cortisol-like molecule is an essential requirement, permitting only selected modifications. Topical activity is favored by substitution of lipophilic groups, which tend to favor local absorption but diminish aqueous solubility. Characteristics of topical agents include fluorine substitution at the 6 and 9 positions, and placement

Dr Mathewson is Professor Emeritus of Anesthesiology and Professor of Respiratory Therapy Education, and Dr Kovac is Associate Professor of Anesthesiology and Medical Director of Respiratory Therapy—University of Kansas Medical Center, Kansas City, Kansas.

Reprints: Hugh S Mathewson MD, University of Kansas Medical Center, 39th and Rainbow, Kansas City KS 66160-7282.

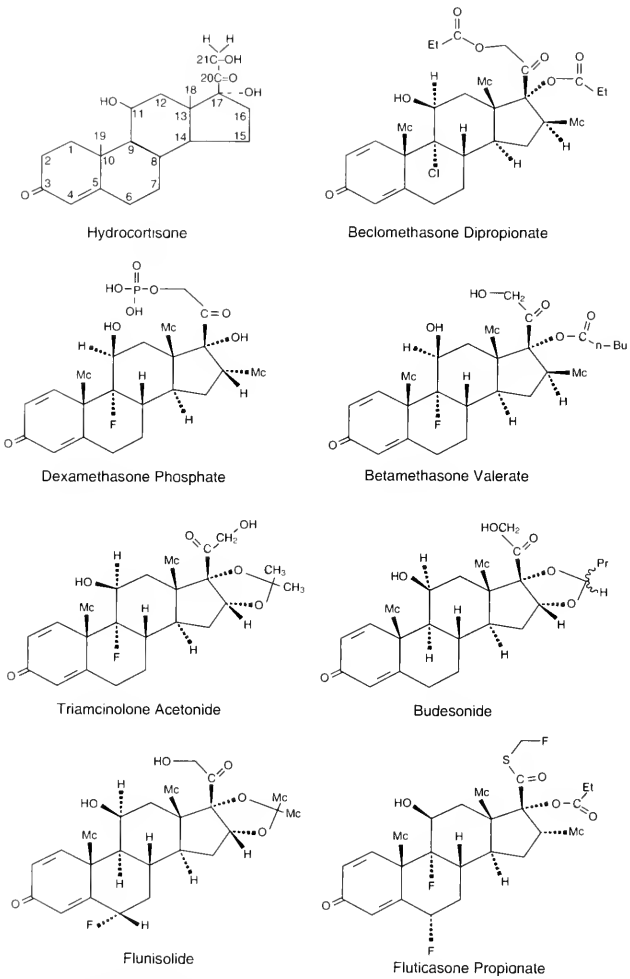

Fig. 1. Structural formulas of corticosteroids suitable for aerosol administration. (The formula for hydrocortisone shows positional numbering.)

of lipophilic groups that are hydrolyzable by the liver in the 16, 17, and 21 positions.

Beclomethasone Dipropionate

Beclomethasone dipropionate,[17] in its original structural form, is a drug closely related to prednisolone and similar to hydrocortisone. Propionic acid esterification in the 17 and 21 positions forms a lipophilic, nearly insoluble, and topically active compound. This drug was introduced in the United States in 1972 as the first corticosteroid to be given exclusively by aerosol. It is still the standard of comparison for new derivatives. Inhaled beclomethasone dipropionate, 400 to 800 μg daily, can reduce the need for oral maintenance steroids in the majority of asthmatic patients who require them in their maintenance regimen. It is about 500 times more potent topically than hydrocortisone or dexamethasone but is ineffective orally due to its low solubility.

The maximum recommended daily dose of beclomethasone dipropionate is 840 μg, which usually does not alter HPA function or produce significant metabolic changes. Increasing the dosage to 2,000 μg daily may provide additional clinical benefit in some patients with refractory bronchospasm but with increased risk of systemic toxicity.[18] There is no evidence that the drug damages the tracheobronchial mucosal lining.[19] It is taken up and biotransformed by the lung tissue so that systemic absorption is minimal. The portion of administered drug that is swallowed is rapidly metabolized by the liver so that little or no cumulative effect occurs.

Triamcinolone Acetonide

The unsubstituted drug, triamcinolone, is a synthetic corticosteroid comparable in potency to methylprednisolone that is marketed for oral use. It is notable for its lack of mineralocorticoid activity, and will actually cause sodium and water diuresis. When conjugated with acetone at the 16, 17 positions (as a ketal), a lipophilic compound is produced that is effective given by aerosol. It is marketed with its own spacer device (Azmacort®). Triamcinolone acetonide shows little detectable systemic activity. Inhalation doses of 400 to 1,400 μg per day were studied in severe steroid-dependent adult asthmatics.[20] A return to normal adrenal function occurred in those patients able to discontinue oral steroids, and side effects were minimal.

Betamethasone Valerate

Betamethasone valerate, unavailable for aerosol use in the United States, is an insoluble ester of betamethasone, a potent corticosteroid related structurally to dexamethasone.

Flunisolide

Flunisolide is an isomer of triamcinolone acetonide. The only structural difference between the two compounds is that a fluorine substituent is in the 6 position in flunisolide and in the 9 position in triamcinolone acetonide. The ketal acetone conjugate in the 16, 17 positions renders flunisolide virtually insoluble in water. The oral aerosol preparation is marketed as a microcrystalline hemihydrate in fluorocarbon propellants, with sorbitan trioleate as a dispersing agent. A study comparing outcomes with flunisolide and beclomethasone dipropionate in 30 steroid-dependent asthmatics did not reveal significant differences.[21]

Budesonide

Budesonide[22] represents a structural departure from other topical steroids. It contains no halogen, and the lipophilic substituent is a 16, 17 butylidine bis-oxy moiety. Although it has been widely used in Europe since 1982,[23] it is not yet available in the United States. It is claimed to have a higher ratio of topical-to-systemic activity than any of the preceding agents.[24] However, a controlled trial did not demonstrate any superiority over beclomethasone dipropionate.[9] Doses of 400 to 2,400 μg per day have been recommended. The higher ranges are more effective,[25] but some adrenal suppression has been reported.[17]

Fluticasone Propionate

Fluticasone propionate is a newly introduced agent of considerable promise, although it has yet to receive the approval of the U.S. Food & Drug Administration. Its structure is significantly different from any of its predecessors. The 17 position is bisubstituted with propionate and fluoromethyl carbothioate groups, both of which can be rapidly hydrolyzed by the liver. Its lipophilic character is reinforced by fluorine substitution at both 6 and 9 positions. The developmental chemistry and relationships to earlier drugs are detailed in a recent article.[11]

Fluticasone propionate has almost twice the topical anti-inflammatory potency of beclomethasone dipropionate.[26] It is not appreciably absorbed from the gastrointestinal tract, but the fraction of active drug absorbed from the lungs after inhalation (and therefore its total systemic availability) has yet to be determined.

The efficacy of inhaled fluticasone propionate, 50 to 2,000 μg/day administered for up to 1 year has been assessed in more than 4,000 patients with asthma.[26] In one clinical study it was found to be as effective at half the dose required for beclomethasone dipropionate or budesonide.[27] Current evidence suggests that it is potentially beneficial and well tolerated in children.[28] In studies on adult asthmatics, fluticasone propionate administered at doses of 1,500 μg/day for 1 year or 2,000 μg/day for 6 weeks did not cause suppression of plasma cortisol levels.[29] In human volunteers, some reduction was noted after intravenous administration of single 2,000-μg doses.[30]

Considering the structural limitations of the glucocorticoid molecule, it is likely that the ideal topical agent will be difficult or impossible to design. However, the widespread adoption of aerosolized steroids as maintenance therapy for mild-to-moderate asthmatics has stimulated research interest. The primary quest is for increased topical potency without toxicity hazards, which could enable clinicians to control severe asthmatic patients without the necessity for oral steroids.[31]

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Choose the single most-correct answer.
The issue and page number of the paper to which the question applies are given in brackets [ ] following the question.

1. Which of the following is ***most correct***?
Increased susceptibility to injury from oxygen toxicity may be associated with
a. young age of the subject (in animals).
b. previous exposure to sublethal concentrations of supplemental oxygen.
c. the presence of certain endotoxins.
d. a generalized decrease in metabolism (as in hypothyroidism).
e. the administration of certain chemotherapeutic agents (eg, bleomycin).
[July 93:739-753]

2. Which of the following is ***most correct***?
Inhaled nitric oxide should
a. reduce intrapulmonary shunt.
b. exert a generalized vasodilator effect on the pulmonary vasculature.
c. selectively decrease the perfusion of ventilated regions of the lung.
d. bind rapidly with hemoglobin to affect the systemic circulation.
e. a & c.
[July 93:754-768]

3. Which of the following is ***most correct***?
Desirable results of hypothermia may include
a. increased $\dot{V}_{CO_2}$.
b. right-shift of oxyhemoglobin dissociation curve.
c. increased blood viscosity.
d. decreased $\dot{V}_{O_2}$ with preserved ability to extract O_2.
e. b & d.
[July 93:769-783]

4. Which of the following statements is ***incorrect***?
a. $\dot{D}_{O_2} = (CI)\,[(1.34)\,(Hb)\,(S_{aO_2}) + (0.003)\,(P_{aO_2})$.
b. $\dot{V}_{O_2} = (CI)\,[C(_{a-\bar{v}})_{O_2}]$.
c. Physiologic supply-dependency refers to a reduction in $\dot{D}_{O_2}$.
d. Pathologic supply-dependency refers to inefficient oxygen extraction.
e. If $\dot{V}_{O_2}$ decreases significantly when $\dot{D}_{O_2}$ is increased, clinical supply-dependency is said to exist.
[July 93:800-828]

5. Which of the following is ***inconsistent*** with the management approach described by Wood and Hall?
a. Clinical evidence of adequate cardiac output includes adequate pulse pressure, warm digits with good nail-bed perfusion, absence of tachycardia, and lactic acidemia.
b. Because the lactic acidosis seen in septic patients with AHRF is always a consequence of anaerobic metabolism, maximizing D_{O_2} will resolve the acidosis.
c. Immediate goal of hemodynamic measurement is the least P_{pcw} to provide an adequate C.O.
d. There is no predetermined target value for P_{pcw} or C.O.
e. None of the above
[July 93:784-799]

6. According to Morris's assessment, which of the following statements is ***incorrect***?
a. Permissive hypercapnia is clearly the superior ventilator management strategy for ARDS.
b. Most medical therapy has never been documented to produce more good than harm.
c. The major advantage of an experimental over an observational study is the strength of causal inference it offers.
d. The randomized double-blind trial is the ideal design for between-group comparison.
e. Therapy standardization by protocol can reduce random and systematic bias introduced by members of the clinical care team.
[July 93:829-841]

7. According to the summary by Dantzker, which of the following is the ***most correct***?
a. Lactate levels can be influenced by acid-base state and nutritional state independent of the adequacy of tissue oxygenation.
b. T_{O_2} provides us with a good estimate of the amount of O_2 actually delivered to the tissues.
c. $\dot{V}_{O_2}$ is easy to assess in the critically ill and is a sensitive index of visceral function.
d. $P_{\bar{v}O_2}$ is an excellent indicator of the adequacy of tissue oxygenation.
e. Above a certain critical level, reductions in T_{O_2} always result in a fall in V_{O_2}.
[July 93:842-846]

8. In the paper by McPeck et al, which of the following is ***most correct***?
   a. Results suggest that 3 of 4 ventilators were able to run the nebulizer to dryness in 20-30 minutes.
   b. Pressures in the nebulizer tubing of 8.5 to 14.7 psig were associated with more efficient nebulization.
   c. Both nebulizers in combination with 3 of the ventilators were able to deliver ≥ 9.6% of the nebulizer charge for the filter.
   d. All of the above
   e. All but c

   [August 93:887-895]

9. In the paper describing a method for predicting expiratory time for a desired intrinsic PEEP level, the following points are made.
   a. Intrinsic PEEP is likely to occur when (1) expiratory time is inadequate for complete exhalation and (2) patients present with premature airway closure.
   b. If the practitioner adjusts a mechanical ventilator to generate applied PEEP ($PEEP_{appl}$) and then progressively shortens expiratory time, at some point $PEEP_I$ and $PEEP_{appl}$ will co-exist.
   c. If R, C, and $PEEP_I$ of the lung are known, t_e necessary to achieve any other $PEEP_I$ (except 0) can be calculated.
   d. All of the above
   e. Only a & b

   [August 93:896-905]

10. In the paper on clinical instructor training, which of the following is ***most correct***?
    a. The importance of interrater reliability in clinical performance evaluation, key performance elements, correct performance, and checklist use was addressed during training of the experimental group.
    b. No difference in interobserver agreement was seen between the experimental and control groups.
    c. The effect size suggests that the intervention had no practical importance.
    d. Instructors with more experience and more advanced credentials performed better than those with lesser experience and lesser credentials.
    e. Videotaping was found to be essential to performance evaluation.

    [August 93:906-915]

11. The 'take home' message from the peak flow meter study by Simmons et al was
    a. bias was observed among flow meters of different brands but not among units of the same brand.
    b. observed bias of none of the devices changed over time.
    c. a subject's apparent PEFR change could be due only to the use of a different PFM.
    d. bias was defined as the constant effect of different operators across all maneuvers.
    e. a & c

    [August 93:916-922]

12. Related to the study by Graybeal & Russell, which of the following is ***incorrect***?
    a. $P_{(a-et)CO_2}$ is small and stable in healthy adults.
    b. In the 80 patients studied (613 paired measurements), correlation between P_{aCO_2} and P_{etCO_2} was positive and < 3% showed disagreement in direction of change.
    c. The authors believe that if P_{etCO_2} is to be an adequate surrogate for P_{aCO_2}, then ΔP_{etCO_2} must reflect ΔP_{aCO_2} in both magnitude and direction.
    d. The frequent changes in V_D/V_T that can occur in critically ill patients may contribute to the poor correlation between P_{etCO_2} and P_{aCO_2} found in the study.
    e. In the authors' review of the literature, no studies were able to retrospectively identify those patients in whom there would be a significant positive correlation between P_{aCO_2} and P_{etCO_2}.

    [August 93:923-928]

13. All of the following statements are correct ***except which one?***
    a. Recognition of changes in pulmonary compliance by bedside measurement may be useful in titrating bronchodilator and surfactant therapy.
    b. Total pulmonary compliance is calculated by dividing the change in pressure by the change in volume.
    c. The commonly held belief that the "educated hand" permits detection of subtle changes in pulmonary compliance has been disputed.
    d. The presence of a large leak renders compliance calculation inaccurate.
    e. Because of a persisting Hering-Breuer reflex, the respiratory muscles of infants are normally relaxed at occlusion volumes above end-expiratory volumes.

    [September 93:985-992]

14. In the case report on ARDS during pregnancy, which of the following is ***most correct***?
    a. The infant's anomalies were directly attributable to the insult sustained by the mother.
    b. ARDS during pregnancy has been attributed to amniotic fluid embolism, eclampsia, placental abruption, and tocolytic therapy.
    c. ARDS is a frequent sequela of acute pyelonephritis in pregnancy (occurring in 25-30% of cases).
    d. The most likely cause of ARDS in the reported patient was polyhydramnios and associated fetal anomalies.
    e. The effect of vasoactive drugs on uterine blood flow is well studied in human subjects.
    [September 93:993-996]

15. According to the paper on occupational asthma (OA), which of the following is ***most correct***?
    a. Prevalence of OA varies little from occupation to occupation.
    b. OA appears to be becoming the most prevalent of all occupational lung diseases.
    c. The mechanism for the development of OA is solely immunologic.
    d. Agents known to be responsible for the excess of cases of OA in respiratory care practitioners are glutaraldehyde, pentamidine, methacholine, and latex.
    e. According to the study by Kern and Frumkin, respiratory care practitioners are more than 10 times more likely than controls to develop asthma.
    [September 93:997-1004]

16. Which of the following statements concerning drug administration during ACLS is ***incorrect***?
    a. A therapeutic option that is inappropriate, without supporting data, and potentially harmful has a Class III designation.
    b. Epinephrine has an alpha-adrenergic receptor-stimulating effect and is given to increase cardiac output.
    c. Excessive catecholamine level can produce tachycardia and recurrent ventricular fibrillation.
    d. When venous access is unattainable, diluted epinephrine, lidocaine, or atropine should be given endotracheally at 2.5 times the given dose.
    e. Administration of sodium bicarbonate facilitates defibrillation and corrects hyperosmolarity and hypernatremia.
    [September 93:1020-1023]

17. Although this study found no differences in the weight of secretions, S_{pO_2}, heart rate, and blood pressure between high-frequency chest-wall compression (HFCC) and manual percussion and drainage (P&D) therapy, several important conclusions can be drawn.
    a. HFCC may not be feasible because of its cost.
    b. Even after more than 500 hours of service, there were no equipment malfunctions with HFCC.
    c. There was a trend toward improved patient perception of comfort with HFCC.
    d. HFCC is less labor-intensive than manual P&D.
    e. All of the above except b
    [October 93:1081-1087]

18. Sulfhemoglobinemia is a rare disorder that
    a. is characterized by peripheral cyanosis and is responsive to 100% oxygen.
    b. is often misdiagnosed as methemoglobinemia because of their spectral proximity.
    c. is probably less common (prevalent) than methemoglobinemia.
    d. should be suspected in the presence of an abnormally high metHb level from a 4-wavelength spectrophotometer without the corroboration of physical signs of metHb.
    e. is described by all of the above except c.
    [October 93:1088-1091]

19. From the study by Akingbola et al, which of the following is ***incorrect***? Use of short-term noninvasive bilevel positive airway pressure
    a. successfully avoided intubation.
    b. reversed atelectasis.
    c. reduced mortality.
    d. may not be an option in confused or combative patients.
    e. has not been rigorously studied in children.
    [October 93:1092-1098]

20. In their study comparing standard capping to a 1-way valve for tracheostomy decannulation, Le et al found that
    a. patients universally preferred the 1-way valve technique.
    b. there was no statistically significant difference between the two methods with respect to time to decannulation or decannulation failure.
    c. thick secretions are not a contraindication to decannulation attempts.
    d. both techniques are labor-intensive.
    e. all of the above are true.
    [November 93:1161-1167]

21. The success of the Respiratory Therapy Consult Service at the Cleveland Clinic reported by Stoller et al
a. required the cooperation and participation of all the medical staff.
b. could not be implemented until the entire respiratory therapy staff had been oriented.
c. resulted in fewer treatments/patient.
d. shows that such a service can be implemented in a large teaching hospital with widespread physician and nursing support.
e. lies in its proven ability to reduce misallocation of respiratory care services.
[November 93:1143-1154]

22. In this TYRS by Brooks and Mishoe, the interpretation of the CT scan requires that the spatial orientation of the patient be
a. caudal to cephalad and left chest is on your right.
b. cephalad to caudal and left chest is on your right.
c. cephalad to caudal and right chest is on your right.
d. caudal to cephalad and right chest is on your right.
e. caudal to cephalad or cephalad to caudal and the left chest is on your right.
[December 93:1201-1204]

23. Within minutes, wood-smoke inhalation causes atelectasis, increased venous admixture, hypoxemia, and pulmonary edema in anesthetized dogs. The addition of 10 cm H_2O PEEP ***did not result*** in
a. restored P_{aO_2} and C_L to pre-injury values.
b. significantly reduced carboxyhemoglobin.
c. reversed pulmonary edema.
d. improved cardiac index and decreased venous admixture.
e. any of the above.
[December 93:1346-1354]

24. The records of 251 infants admitted to an NICU were retrospectively reviewed to determine the relative contribution of various treatments on surfactant and central venous catheters (CVCs) and dexamethasone therapy to the incidence of neonatal infection. They found that
a. cultures were taken more often on cases than controls in the dexamethasone- and surfactant-treated series.
b. cases in the central-line group had significantly more positive blood cultures than their matched controls.
c. birthweight, gestation, and CVC were significantly associated with neonatal sepsis.
d. neither surfactant nor dexamethasone treatment was significantly associated with the incidence of neonatal infection.
e. all of the above are true except c.
[December 93:1355-1363]

25. The use of β-adrenergic bronchodilators and antimuscarinics in combination by inhalation is being investigated for a number of airway disorders. The evidence supports that the effect(s) of this combination is/are
a. stronger than either agent alone in asthmatics with acute and stable disease.
b. stronger if the antimuscarinic is administered first.
c. stronger and more prolonged than either agent alone in patients with stable COPD.
d. stronger than either agent alone in patients with chronic bronchitis.
e. all of the above.
[December 93:1364-1388]

26. All of the following ***except one*** are common characteristics of β-adrenergic bronchodilators. ***Which one?***
a. They stimulate the receptors of the sympathetic neural pathway of the central nervous system.
b. They have a significant effect on bronchiolar smooth muscle tone even though there are no sympathetic fibers in the lung.
c. They stimulate the conversion of cATP to cAMP as long as adenyl-cyclase levels are low.
d. Their main therapeutic effect is bronchodilatation.
e. They inhibit antigen-induced release of mast-cell mediators.
[December 93:1364-1388]

27. A woman sustained blunt trauma to her abdomen as the result of an automobile accident. The diagnosis of diaphragmatic rupture was made from the initial chest radiograph that showed
a. deviation of the trachea toward the affected side.
b. a widened mediastinum.
c. the nasogastric tube and the outline of the stomach and bowel loops in the chest.
d. all of the above.
e. a & c only.
[December 93:1423-1427]

28. Which of the following statements about meta-analysis (MA) is true?
a. MA is a qualitative integration of the findings of a number of studies.
b. The consensus of clinicians and investigators is that only papers published in peer reviewed journals should be included in a MA.
c. The probability of rejecting a false null hypothesis (ie, power) is reduced by MA.
d. If a MA is said to be stable to additional studies, a small number of studies with insignificant results will reduce a significant effect size to insignificance.
e. None of the above
[January 94:34-49]

29. According to studies cited in the paper by Heffner, portable chest radiographs are indicated in critically ill patients ***except***
a. after placement of a chest tube.
b. after most tracheotomies.
c. after most translaryngeal intubations.
d. on admission to the ICU.
e. with changes in clinical status suggesting radiographically demonstrable disorders.
[January 94:51-62]

30. Which of the following is ***incorrect***?
a. The normal result of inhaling CO_2 is an increase in central neural drive and level of ventilation.
b. With the progressive hyperoxic hypercapnic technique, every 10 torr increase in P_{CO_2} is expected to increase minute ventilation by 20 L.
c. During exercise, a normal subject increases minute ventilation by increasing tidal volume and breathing rate.
d. A crushed phrenic nerve will repair itself in 2-6 months.
e. None of the above is incorrect.
[January 94:67-69]

31. According to the review by Hess, which of the following is ***incorrect***?
a. The appropriate dose of bronchodilator via MDI for intubated patients is unresolved.
b. With high-dose β-agonist therapy, the patient should be monitored for side effects such as arrhythmias, tremor, and hypokalemia.
c. During passive mechanical ventilation, WOB can be estimated from measurements of pressure and flow at the proximal airway.
d. Peak inspiratory pressure and auto-PEEP were reduced after bronchodilator administration in the series by Gay et al.
e. The presence of a humidifier in the ventilator circuit is desirable during therapeutic aerosol administration.
[February 94:105-122]

32. According to Durbin's review, which is ***incorrect***?
a. Changes in $S_{\bar{v}O_2}$ can be interpreted independently without consideration of other patient variables.
b. Clinical efficacy is defined as the ability of a device or treatment to achieve its ascribed goals under controlled circumstances.
c. Clinical effectiveness is the ability of a device or treatment to achieve the desired results in clinical applications that cannot be rigorously controlled.
d. Cost analysis should include consideration of direct costs, indirect costs and savings, and caregiver impact.
e. A monitor is cost-effective when it costs less or incurs less risk than other monitors in use—in addition to being clinically effective.
[February 94:123-136]

33. According to the article on flow-triggering systems, which of the following is ***incorrect***?
a. Sassoon has shown that in the Puritan-Bennett 7200ae, WOB is less during flow-triggering than during pressure-triggering.
b. On certain ventilators, the Percent Rise Time and Pressure Slope settings control how quickly the limit variable is reached during inspiration.
c. The fastest rise-time always results in the lowest WOB.
d. Flow-triggering appears to improve patient-ventilator synchrony.
e. Flow-triggering cannot reduce the WOB due to the endotracheal tube or auto-PEEP.
[February 94:138-144]

34. According to the review by Judson and Sahn,
a. the rate of ciliary beat and the proportion of ciliated cells increase distally in the airways.
b. tracheobronchial suction can impair mucociliary clearance.
c. tracheal intubation almost always leads to decreased mucus secretion.
d. Sackner et al's work suggests that mucus transport is not impaired as long as oxygen concentration is maintained below 75%.
e. the clinical effectiveness of β-agonists in mobilizing secretions has been well established by published studies.
[March 94:213-226]

35. According to the review by Campbell, which is ***incorrect***?
a. 'Across the board,' the more ventilator alarms that are operative, the safer is the patient.
b. Although it is technically difficult, pressure measurement at the distal end of the endotracheal tube is optimal.
c. The major factor affecting the volume that reaches the patient's lungs through an intact circuit is probably the compressible volume of the circuit.
d. Unstable mechanically ventilated patients may require constant or almost constant surveillance.
e. Ventilator circuits become contaminated quickly (within 8 hours).
[March 94:227-236]

36. According to the paper by Graybeal and Russell on end-tidal carbon dioxide measurement, which is ***incorrect***?
a. $P_{(a-et)CO_2}$ has been shown to correlate with alveolar dead space and dead-space-to-tidal volume ratio.
b. The design of the Raman spectrometer utilizes the light-scattering properties of gases.
c. No significant differences were found between the measurements by Raman and mass spectrometry.
d. An advantage of the Raman spectrometer is the fact that a measurement error in gas present in a mixture does not affect the reported values of other gases present in the mixture.
e. The results of the reported study suggest that for P_{etCO_2} it may be desirable to use the same device for all measurements on a given patient.
[March 94:190-194]

37. In his conference summary, Dr Pierson emphasizes
a. that respiratory care practitioners need to become less tied to equipment and more committed to becoming educators, consultants, and coordinators of care.
b. that the more we are able to care for our patients at home, the more we enhance their quality of life and reduce the cost of their care.
c. that all patients who fail to wean from mechanical ventilation in the ICU are candidates for long-term ventilation.
d. a & b only.
e. all of the above except b.
[April 94:294-308]

38. Patrick Dunne calls for modernization of our outdated system of reimbursement for home respiratory care. The impetus for this modernization ***does not include*** which of the following?
a. Only the economically disadvantaged can be cared for at home
b. The provision of respiratory care services in the home has been proven to be cost-effective.
c. The contribution of the respiratory therapist in home health-care is being recognized.
d. The home may become the primary health-care site of the future.
e. The evidence supports that with proper education, training, and follow-up, medically naive caregivers can collaborate with home health-care professionals to achieve successful patient outcomes.
[April 94:309-320]

39. Fletcher reports that NOD ***cannot be*** reliably predicted by
a. daytime P_{aO_2}.
b. mean nocturnal saturation.
c. episodes of desaturation during REM sleep defined by polysomnography.
d. a & b only.
e. age and gender.
[April 94:333-346]

40. According to Giordano, several important factors limit the function of RCPs in the home. Which of the following is/are true?
    a. Respiratory home care can only be provided by RCPs to patients who can pay for it out-of-pocket. Because RCPs cannot be reimbursed for home care, patients at home with respiratory disease are discriminated against.
    b. There are no data to show the cost-effectiveness of RCPs in home care.
    c. Only a few respiratory therapists have a strong desire to work in the home-care setting.
    d. RCPs must expand their education and vision to be ready to practice in patients' homes when the opportunity arises.
    e. All of the above are true.
    [April 94:321-332]

41. Stoller reports that the best predictor of hypoxemia at altitude (cabin pressure = P_B at 8,000 ft) is
    a. P_{aO_2} at sea level % predicted FEV_1.
    b. P_{AO_2} at sea level.
    c. $D_{(a-A)O_2}$ at sea level.
    d. MMEF < 100 L/min.
    e. all of the above.
    [April 94:347-362]

42. Unlike center-based sleep studies, those done in the patients' homes often
    a. are performed by untrained personnel.
    b. lack technical standardization.
    c. are unattended.
    d. may include all of the above.
    e. must be confirmed by center-based studies.
    [April 94:390-400]

43. An abnormality in any component of the mucociliary escalator—the gel and sol layers and the cilia themselves—can result in
    a. ineffective cough.
    b. bronchiectasis.
    c. infection.
    d. all of the above.
    e. a & c only.
    [May 94:440-455]

44. The principles that apply to exercise training in general
    a. are that exercise programs should be designed to achieve specific goals in the target muscle group; the effect of training the muscles is directly related to the intensity (magnitude) and duration of the load applied to the muscles; and the effect achieved by muscle training is lost if the exercise is stopped.
    b. apply to all voluntary skeletal muscles except those of respiration.
    c. apply to all voluntary skeletal muscles including those of respiration.
    d. are a & b only.
    e. are a & c only.
    [May 94:481-500]

45. Noninvasive positive pressure ventilation is clearly indicated for patients with
    a. chest-wall disorders such as kyphoscoliosis and the deforming sequelae of tuberculosis.
    b. neuromuscular disorders and limited swallowing capability.
    c. any patient with hypercapnia.
    d. a & c only.
    e. b & c only.
    [May 94:501-514]

46. Any individual who requires ventilatory support can be weaned from the endotracheal-tracheostomy tube and ventilated noninvasively ***except***
    a. those who cannot tolerate heavy sedation.
    b. those whose bulbar muscle function is inadequate for swallowing.
    c. those who require even minimal supplemental oxygen.
    d. those whose peak cough expiratory flows are < 50 L/min.
    e. all of the above.
    [May 94:515-531]

47. In the study comparing the work of breathing associated with artificial airways in a lung model, Davis et al found
    a. that as flow increased, measured work of breathing increased and peak negative pressure decreased.
    b. that measured work of breathing varied inversely with endotracheal tube size.
    c. that in vitro measurements of airway resistance grossly overestimate those made in vivo.
    d. that the resistance to airflow in tracheostomy tubes is significantly higher than that in endotracheal tubes.
    e. all of the above.
    [June 94:611-616]

48. Vocal cord paralysis in newborn infants
    a. occurs in 23% of all live births.
    b. is idiopathic in about a third of patients.
    c. is only incident in full-term deliveries.
    d. can only be confirmed diagnostically by rigid bronchoscopy.
    e. should be suspected in any infant born prematurely.
    [June 94:630-634]

49. Using the Siemens 900C ventilator, Madsen et al tested the expiratory hold and the clamping/transducing methods for quantitating intrinsic PEEP and
    a. found that the values produced by both methods were in good agreement.
    b. discovered that circuit compliance did not influence the measurements of intrinsic PEEP with either method.
    c. concluded that the clamping/transducing method was more feasible for bedside use than the expiratory hold method because it was less time consuming.
    d. confirmed that these measurements were grossly inaccurate in patients who were pharmacologically paralyzed.
    e. discovered all of the above.
    [June 94:623-626]

50. In their article on cerebral arterial gas embolism (CAGE), Layon, Layon, and Nelson emphasize
    a. that the risk of misdiagnosis due to the body's slow response to dysbarism.
    b. that the diagnosis must be based on sophisticated laboratory data because information from the history and physical are often misleading.
    c. that the extent of the neurologic morbidity is directly related to the volume and duration of air in the cerebral blood volume.
    d. that, overall, CABG is fatal in about 50% of cases.
    e. all of the above except c.
    [June 94:635-644]

RETROSPECTROSCOPE REDUX:

Who's Who in World History

A few nights ago I watched a rerun on television of one of Groucho Marx's quiz shows, "You Bet Your Life." Groucho's standard format is first to interview a pair of contestants, slyly insulting each in his inimitable way, and then giving them a chance to make a little money. All they must do is answer correctly four questions, in a category they themselves have selected. The first round of questions is pretty easy and most contestants win a few hundred dollars and so qualify for a chance at the big money, either $2,000, $5,000, or $10,000, depending on the turn of a wheel. Now the contestants must provide the right answer to a single but not-so-easy question. This time the question was, for me, elementary—"Who discovered penicillin?" The contestants hadn't the foggiest idea and left the stage without the big prize and dejected.

I wondered and kept on wondering why they didn't know. Certainly the discovery of penicillin has resulted in saving more lives than most any other single event since the beginnings of history. I turned to the F's in an unabridged dictionary[1] to see if Fleming's name was there; it was indeed—"Sir Alexander, Scottish bacteriologist and physician; co-discoverer of penicillin, 1929." I then turned to the same dictionary's "Major Dates in World History," a special supplement of 21 oversize pages containing, in all, 1,770 entries. The first entry was 3200 B.C. (the first dynasty in Egypt) and the last was Pope Paul VI's ecumenical council at the Vatican in 1965.

I found the year 1929, when Fleming discovered penicillin. There were four entries for 1929: the reign of Zogu (King of Albania), the acceptance of the Young reparations plan by the Germans and the Allies, the crash of the New York Stock Market, and the inauguration of Herbert Hoover as President. The discovery of penicillin was not there. I wondered whether Roentgen's 1895 discovery of x-rays was a "major date in world history"; it was not, though the entries for 1895 included the Sino-Japanese War, the Dreyfus case, the reign of Nicholas II of Russia, and "Far Eastern Crises." Surely, I mused, Harvey's discovery of the circulation of the blood must have made it. But although the year 1628 included the reign of Philip IV of Spain, the reign of Charles I of England, the signing of the Petition of Right by Charles I, and the chartering of the Massachusetts Bay Company, it did not include William Harvey.

I then thought that it would be interesting to see whether any of the 56 discoveries honored by Nobel prize awards in medicine or physiology between 1901 and 1965 (the dates of the first Nobel award and the compilation of Random House's list) were included in "Major Dates in World History." I didn't expect to find Crick, Watson, and Wilkins's discovery of the double helical structure of DNA (too esoteric) but I thought I might find Von Behring (who discovered diphtheria antitoxin), or Ross (who found that malaria was transmitted by mosquitoes), or Koch (who identified the tubercle bacillus as the cause of tuberculosis), or Banting and MacLeod (who extracted insulin from the pancreas and initiated the treatment of diabetes), or Domagk (who inaugurated modern chemotherapy with the discovery of Prontosil®, forerunner of sulfanilamide), or Theiler (who prepared vaccines effective against yellow fever), or Enders, Weller, and Robbins (who made polio vaccine possible). No; none made the list. Neither did any pre-Nobel biomedical scientist such as Claude Bernard, Gregor Mendel, Louis Pasteur, Edward Jenner, or Charles Darwin.

With the single exception of the discovery of "blue galaxies" in 1960, physics, chemistry, astronomy, and mathematics also struck out—not a single line for Aristotle, Copernicus, Galileo, Kepler, Newton, Cavendish, Lavoisier, Volta, Dalton, Thomson, Hertz, Curie, Einstein, Bohr, or Lawrence, and not a single word about electricity, atomic structure, or radioactivity.

Engineering and invention fared better. Lucius

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Tarquinius Priscus (616-578 B.C.) made the list for building the "great sewer" (Cloaca Maxima). Prince Chen (259-210 B.C.) got in by standardizing weights and measures. The Tennessee Valley Authority (1933) is there, as is the use of pilotless jet missiles (1944), the dropping of the first atomic bomb (1945), the launching of Sputnik (1957), and the U-2 high altitude reconnaissance plane (1960). Indeed, the atomic bomb and its more powerful derivatives received 11 separate entries and space vehicles racked up 15 (7 more than all of the earlier explorers of the then-unknown world—Ericson, Columbus, da Gama, Vespucci, de Leon, Drake, Raleigh, and Hudson). But invention and engineering completely ignored the printing press, the steam engine, the airplane, radio, television, and radar; it even skipped the whole Industrial Revolution.

Art? Music? Painting? Architecture? Literature? No—not even Homer or Leonardo or Shakespeare. Only three of the 1,770 entries mention a museum (Ptolemy's in Alexandria about 300 B.C.), a library (Pope Nicholas V's in the Vatican about 1450), or a book (the King James version of the Bible in 1611).

Law made it 5 times for legal, judicial, or constitutional reforms: Hammurabi, Babylon, eighteenth century B.C.; Solon, Greece, about 590 B.C.; Roman law, 451 B.C.; Magna Carta, 1215 A.D.; and systemization of English law, about 1280 A.D.

Social reform had 3 entries: prohibition of slavery in British colonies (1833), restrictions on child labor in England (1834), and Lincoln's Emancipation Proclamation (1863).

Because these account for only 50 of the 1,770 entries, I began to wonder "What *is* history? What were the other 1,720 entries?" I haven't categorized and tallied them but most of them deal with who ruled whom, when, and how; how rulers met their death or meted it out to others (beheading—old style, guillotine—modern style, assassination, poisoning, suicide); invasions, wars, battles, massacres, and the number of casualties; treaties and when they were broken; burning, sacking, razing, looting, inquisition, and excommunication. (This isn't much different from Ambrose Bierce's definition of history: "An account mostly false, of events mostly unimportant, which are brought about by rulers mostly knaves, and soldiers mostly fools."[2])

Back to Groucho Marx and "Who discovered penicillin?" If the Random House supplement is representative of modern teaching of world history, Groucho's contestants didn't really have a chance at their $10,000. *Is* it representative? I tracked down about 10 history texts now in use in our high schools and colleges and leafed through them. Some, I'm glad to say, do devote a fair number of pages to the arts and sciences; one even traces science from its beginnings in Greece (great science) through Rome (little science), to the intellectual revolution of the 17th and 18th centuries and the new intellectual revolution of the 19th and 20th centuries, and it does indeed mention Banting, Salk, Sabin, Waksman, Fleming, and Domagk—right in the middle of a history text! Others give only a nod to the arts and sciences. Most treat art and science in separate chapters—not really part of the real ongoing history of kings, princes, czars, emperors, presidents, popes, wars, and treaties, and easy to omit if the instructor chooses to skip about.

One interesting presentation was not in a history text but in JB Conant's *Harvard Case Histories in Experimental Science*.[3] Conant was an eminent chemist and educator who was President of Harvard University between 1933 and 1953. He believed it important for future voters and those they elect to office to understand the "tactics and strategy" of science and how science fits in with all other fields of human activity. His method was to show when, how, and why great scientific discoveries came about.

His Chapter Two deals with the great Chemical Revolution of 1775-1789 (ushered in by the discovery of oxygen), and he intertwines its events with those of three other revolutions: the Industrial Revolution, the Declaration of American Independence, and the French Revolution. Here, slightly abridged, is *his* chronological table of what mattered between 1760 and 1799 (the starred events were also listed in Random House's supplement; the others were not). [See table on page 852.]

Conant might have gone beyond interweaving four revolutions into one chronological table; he might, from his vantage point almost two centuries later, have analyzed critically the influence of each on the quality of life throughout the world in mid-20th century. The American Revolution might have received first place with the Industrial and Chemical Revolutions (and the Revolution in Physics that followed just a few years later) close behind. Two hundred years from now, the order may be reversed. Or the Revolution in Biomedical Sciences may take top place. In any case, I fervently hope that Random House, if space prevents *additions* to its "Major Dates in World History," will be willing to delete "Reign of Zogu, King of Albania" from its 1929 listings and substitute "Fleming discovers penicillin."

JULIUS H. COMROE, JR.

References

1. The Random House dictionary of the English language, unabridged edition. New York: Random House, 1966.
2. Bierce A. The Devil's dictionary. New York: Dover Publications, 1958 (republication; original publication 1906).
3. Conant JB. Harvard case histories in experimental science. Cambridge: Harvard University Press, 1957 (Volume 1, pp. 113-114).

CONANT'S CHRONOLOGICAL TABLE

1760	Smeaton improves blast furnaces for producing cast iron.
1760	Bridgewater canal completed; halves cost of coal in Manchester.
1760	Factory system and use of water-driven machinery firmly established in English silk industry.
*1765	Stamp Act
1766	Cavendish (London) isolates and describes hydrogen gas (inflammable air).
1769	Watt's first steam engine.
1772	Lavoisier's experiments with sulfur and phosphorus. Priestley publishes his test for the "goodness" of air.
1774	Death of Louis XV.
1775	Lavoisier's "Easter Memoir" on calcination. ["Calcination" here refers to the heating of metallic mercury in air to yield mercuric oxide; the latter, heated to much higher temperatures, decomposes into metallic mercury and oxygen gas.] Priestley's effective discovery of oxygen.
*1776	Declaration of American Independence.
1776	Publication of Adam Smith's *Wealth of Nations.*
1778	Lavoisier's revised memoir on calcination [and discovery of oxygen].
*1781	Capitulation of British at Yorktown.
1782 to 1783	Composition of water established; spread of Lavoisier's ideas; last stand of phlogiston theory.
1783	Lavoisier and Laplace determine large number of specific heats.
1784	Cort's pudding process for making malleable iron from cast iron using coal as fuel.
*1783	Peace between Great Britain and the United States.
1784	Watt's improved steam engine.
1785	Watt proposes bleaching textiles with chlorine gas.
*1787	Constitutional convention at Philadelphia.
1789	Publication of Lavoisier's *Traité Élémentaire de Chimie* setting forth results of the chemical revolution in clear and systematic form.
*1789	Fall of the Bastille (July).
1791	Birmingham mob burns Priestley's house.
*1792	Attack on the Tuileries, Louis XVI a prisoner; French Republic proclaimed.
*1793	War between Great Britain and France, continues with only short truces until 1815.
1794	Execution of Lavoisier (May 8); fall and death of Robespierre (July); end of [the Reign of] Terror.
1796	Production of cast iron in Great Britain 125,000 tons, double the figure of a decade earlier.
*1798	Battle of the Nile. Nelson's victory ensures supremacy of British fleet.
1798	Publication of *Essay on Population* by Malthus.
*1799	Napoleon becomes First Consul.

Books, Films, Tapes, & Software

Comprehensive Perinatal and Pediatric Respiratory Care, by Kent Whitaker BS RRT, with 4 contributors. Soft-cover, 688 pages, illustrated. Albany NY: Delmar Publishers, 1992. $35.95. 1-800-347-7707.

Kent Whitaker, a respiratory therapy instructor at Weber State University in Ogden, Utah, has enlisted several specialist contributors to round out this book, designed in part to be used as a study guide for students taking the NBRC exam. These specialists (a home care consultant, a pharmacy director, a nursing professor, and a neonatal-perinatal physician) contribute to the 5 sections: Development and Care of the Fetus— Conception to Birth, Care of the Neonatal and Pediatric Patient, Causes and Care of Illness in Perinatal and Pediatric Patients, Management of Ventilation and Oxygenation, and Transport, Home Care, and Care of the Parents.

Simple diagrams, radiographs, photos, and tables are provided to help with explanations; and at the end of each chapter, a bibliography and suggested reading list are found. Each chapter also contains a self-evaluation posttest, with answers provided in the back of the book.

Comprehensive Perinatal and Pediatric Respiratory Care details care of the newborn in 20 chapters, with several chapters addressing different topics under each of the above sections. I find the chapters to be easy reading; however, only the most basic information is covered. It appears to be a good resource book for a student who has no previous knowledge of neonatal medicine. The author addresses step by step the details necessary in performing particular clinical functions. For example, in Chapter 7, Respiratory Care Procedures, he details suctioning, giving a brief description of indications for suctioning, as well as information about the equipment, procedure, and hazards of suctioning. It is written in a clear, precise format, is readable, and allows for easy interpretation. For respiratory therapists working in a Level-1 nursery, this book is a good reference. The author has included excellent illustrations with lots of photos and drawings of how equipment should be set up, again making for ease of understanding. He gives brief synopses of some newer technologies that are quite commonplace in a more advanced nursery setting (ie, surfactant, high-frequency ventilation, and ECMO).

In addition to a glossary and index, a practice exam is provided that closely resembles the NBRC exam. Whitaker not only gives the reader the answers to his questions, but he also follows them up with the rationale for his thinking and references the chapter in which information can be found.

Overall, I consider this book to be beneficial to a student in a respiratory therapy program learning for the very first time about the care and treatment of neonates.

Brenda Kaye BA RRT
Research Assistant
Neonatal/Pulmonary Research Group
Department of Respiratory Care
Children's Hospital
Oakland, California

Medical Terminology Simplified: A Programmed Learning Approach by Body Systems, by Barbara A Gylys MEd CMA-A. Soft-cover, illustrated, 492 pages, with 2 audiotapes (4 sides). Philadelphia: FA Davis Co, 1993. $29.95.

This text is written to provide students with an easier and more practical method with which to learn medical terminology. We believe that it is better than other similar books because it employs techniques other than rote memorization to teach the vocabulary. Included with the definitions are explanations of how each word is formed, as well as the origin of each word, which may help the student with subsequent terms. This book includes excellent four-color, three-color, and black and white illustrations, flash cards, and accompanying audiotapes to assist the student with review, reference, and pronunciation. The text is also unique because it includes appendices consisting of both a medical-word and an English-term index; clinical, radiographic, and laboratory procedures that are commonly used to diagnose and treat patients; medications; abbreviations; and a summary and description of medical specialties. Interestingly, pulmonology *is not* included in the author's list of medical specialties. The text is comprehensive and adequately covers each body system.

Medical Terminology Simplified is intended for college-level allied health or nursing students. Although the text would probably be used for science and health career majors, it is written so that even an individual who had never been exposed to medical terminology could gain a basic understanding of medical vocabulary. There is much repetition at the book's beginning, with the frames becoming progressively more challenging.

This book is designed to teach students medical terminology through a process called word-building. It can be used either as a self-instruction manual or with an instructor. The text is prefaced by learning objectives and pronunciation guidelines, is composed of 10 units, followed by 7 appendices, and a set of flash cards. Twenty full-color plates of each body system are included at the beginning of the book, and three-color and black and white illustrations are interspersed throughout the text. Unit 1 introduces the student to the setup and instructions that will be required to efficiently master subsequent units. Instruction is clear and easy to understand. Each of the 10 units is composed of several frames. Students are instructed to cover the answers to fill-in-the-blank statements with a sliding card (which accompanies the text), removing it as they progress through the unit. This method provides students with immediate feedback. Pronunciation keys for each medical term are included in the frame answer boxes. Unit 1 also outlines the basic elements of medical words for the learner, including prefix, suffix, word root, and combining form. Each section in a unit ends with a matching review and answer key. Units end with a case study, accompanying audiocassette exercise (to assist with pronunciation and spelling), and "Unit Exercises" (definitions, vocabulary, and anatomic activity). Because there are 10 units, this book is well suited for students enrolled in a 10-week medical terminology course. The appendices are available for reference and review. Perforated flash cards are a nice touch.

The book is easy to understand and to read. The use of immediate feedback is a proven learning aid. The use of case studies lends a practical, relevant aspect to the text.

Illustrations are clear, colorful, accurate, and nicely labeled. Instructions throughout the text are easy to read and understand, and the index is complete.

Appendices are useful, easy to find, and readable. Reinforcement and referencing frames are provided at the end of each review answer key in the event that the student is not satisfied with his or her performance. These are well-organized and easy to use.

Also available is an Instructor's Manual, that provides a course outline, crossword puzzles, sample tests, and transparencies. Also included in either WordPerfect or ACSII format is a complete test bank. For those interested in enhanced self-instruction, a color computer program is available for use on IBM-compatible machines.

Medical Terminology Simplified is a useful and comprehensive text designed for the college-level student. It may be used as part of a course or as a self-instruction manual. The book is easy to read and understand and provides much opportunity for learning and reinforcement not only within the text but also through the use of case studies, flash cards, audio cassetes, and appendices.

Crystal L Dunlevy EdD RRT
Assistant Professor &
Director of Clinical Education

Seema Parekh CRTT
Senior Student
Respiratory Therapy Division

The Ohio State University
Columbus, Ohio

The Diagnosis and Management of Paediatric Respiratory Disease, by Robert Dinwiddie, with 5 contributors. Hardcover, 299 pages, illustrated. New York: Churchill Livingstone, 1990. $45.00.

Dr Dinwiddie, a consultant paediatrician from London, is very thorough and detailed in his descriptions of pediatric respiratory diseases, using many radiographs, photos, and diagrams to help the reader understand. In addition, he offers many examples of disease incidence rates in the United Kingdom and the United States. All chapters are well referenced and many include sources of additional information for those interested.

The book consists of 14 chapters, beginning with the development of the lungs and physiology of breathing. The author works his way through birth and the neonatal period, and then into a variety of pediatric diseases (such as respiratory tract infections, asthma, aspiration syndromes, pulmonary tuberculosis, and ear, nose, and throat problems). Each of his chapters is succinct and flows easily thru a series of events. He is very thorough in his details, giving a nice overview of the disease and how to treat it. Generally, this description encompasses pathogenesis and clinical features, and often includes management, complications, and other problems. His chapter on cystic fibrosis is an example; it is excellent and very detailed. I found the whole chapter to be interesting to read, as were most of the chapters of his book.

This book does not necessarily detail the respiratory care aspects of pediatric respiratory diseases. It should be used as a reference book to obtain the history and background of different pediatric diseases, but correlating the information provided with current treatment modalities could result in a better understanding of why we are doing what we are doing.

Brenda Kaye BA RRT
Research Assistant
Neonatal/Pulmonary Research Group
Department of Respiratory Care
Children's Hospital
Oakland, Californi

Neonatal and Pediatric Respiratory Care, 2nd edition, edited by Patricia Beck Koff MEd RRT, Donald Eitzman MD, and Josef Neu MD. Hardcover, illustrated, 509 pages. St Louis: Mosby-Year Book Inc, 1993. $41.95.

The second edition of **Neonatal and Pediatric Respiratory Care** reflects the changes and advances made since the first edition was published 5 years ago. It is a comprehensive, solid text, intended for serious students of neonatal and pediatric respiratory care and should appeal to respiratory care practitioners, nurses, and physicians who specialize or intend to specialize in this area. It offers an excellent resource of pathophysiology and treatment as well as practical technical guidance.

The first two chapters, "Development of the Cardiopulmonary System" and "Physiologic Development," are very detailed and thorough and offer the necessary foundation for understanding neonatal respiratory care. The next two chapters, "Patient Assessment" and "Radiographic Evaluation," are excellent chapters to prepare the clinician for competent patient assessment. "Radiographic Evaluation" is probably my favorite chapter of the entire text. The author combines excellent descriptions and illustrations of chest radiographs and their associated diagnoses, and provides good practical advice for the clinician. The chapters covering diseases are comprehensive, from neonatal and pediatric parenchymal diseases to obstructive airway diseases in infants and children. The chapter "Surgical Lesions of Pediatric Airways and Lungs" is well illustrated. In the chapter entitled "Oxygen Therapy," the author includes a discussion on "Hyperbaric Oxygen Therapy" (HBO). Because the uses and benefits of HBO continue to be controversial, I am surprised at the author's strong positive statements on outcomes on Page 244 without adequate references. The chapters covering mechanical ventilation have been expanded to include recent information on high frequency ventilation. The chapter on surfactant replacement therapy is excellent and complete, covering surfactant composition and unresolved issues not answered yet in clinical trials. The chapter on pediatric home care is very good, outlining the discharge process and home therapy. The remaining chapters cover congenital heart disease, apnea of prematurity, sudden infant death syndrome, neurological and neuromuscular impairments, ARDS in children, pharmacology, CPAP, arterial blood gases, airway care and chest physiotherapy, transport, and developmental outcomes.

Overall, this text is best described as both excellent and comprehensive. I found the book pleasantly easy to read, in spite of the depth and detail of the text. The editors have maintained good consistency in form and style, which is admirable considering there were 39 contributors. I liked the format of having "reader objectives" written at the beginning of each chapter—a helpful tool to organize thoughts. A generous number of illustrations are provided, and most are excellent; however, I found the use of tables excessive, to the point of being somewhat tedious. I do not really consider this a fault because I recognize the importance of the material presented and am not able to offer an alternative because a great deal of information included in the book lends itself to tabular form. Finally, I consider the book a real value. The cost, $41.95, is reasonable for such a comprehensive, quality text.

Theresa Reno BA RRT
Director
Department of Respiratory Care
Bowie Memorial Hospital
Bowie, Texas

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Calendar of Events

AARC & AFFILIATES

August 17-19 in Atlantic Beach, North Carolina. The NCSRC hosts its 16th Annual Symposium, "Clinical Technology: Accessing Our Future," at the Sheraton Resort. Contact Beverly Dukes (704) 342-6795 or Debby Harlan (704) 379-5889.

August 25-26 in Jacksonville, Florida. The FSRC announces their 12th Annual River City Symposium at the Marina Hotel. Topics include patient-focused care, continuous quality improvement, and TB/HIV and health care workers; activities include a riverboat cruise. Participants can receive up to 12 CRCE credit hours; the preregistration deadline was Aug 10. Write River City Symposium, Cardiopulmonary Department, 4301 Belfort Rd, Jacksonville FL 32216. (904) 296-3716.

September 1-2 in Tucson, Arizona. The ASRC announces their fall educational seminar. The seminar, with the theme "Shoot for the Stars," emphasizes the RCP's place in the health care structure of the future. Contact Leanna Reece, Program Director, Pima Medical Institute, 3350 E Grant, Tucson AZ 85716. (602) 326-1600.

September 13 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the fifth of a 6-part videoconference series: "Professor's Rounds in Respiratory Care: Hospital Operational Restructuring and Respiratory Care" (1 CRCE credit). For information, call (214) 830-0061.

September 14-16 in Ocean City, Maryland. The Maryland/District of Columbia Society of Respiratory Care presents the 13th Annual Conference by the Sea, at the Carousel Hotel. The symposium highlights reengineering, benchmarking, and therapist-driven protocols. Contact Elgloria Harrison RRT at (703) 497-2500, or Sharyon Brown CRTT at (202) 884-3125.

September 15-16 in Bethel, Maine. The MSRC presents its annual fall seminar at the Sunday River Resort. Topics include NIH asthma treatment/management guidelines, the RCP's future role in health care, pediatric BiPAP, case reviews, and health care promotion. A barbecue, dance, vendor reception, and golf tournament are planned. Participants can receive up to 10 CRCE credit hours. Contact Garry Michaud RRT, Respiratory Care Department, Penobscot Bay Medical Center, Glen Cove, Rockland ME 04841. (207) 596-8472.

September 22-23 in Napa, California. Chapter 10 of the CSRC, the American Lung Association of the Redwood Empire, and the Respiratory Therapy Program at Napa Valley College present the 12th Annual Napa Valley Conference, "Current Concepts in Cardiopulmonary Care." Contact Kate Benscoter at (707) 253-3145.

September 24-27 in San Juan, Puerto Rico. The Puerto Rico Society for Respiratory Care (PRSRC) hosts the Third Latin American Congress for Respiratory Care at the San Juan Convention Center. Featured presentations cover all aspects of critical care and pulmonary rehabilitation. Contact PRSRC President Norma Cruz-Hiraldo at (809) 764-5788.

September 27-28 in Honolulu, Hawaii. The HSRC announces its 21st Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Contact Helen Ono RRT, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532, fax (808) 547-9535.

OTHER MEETINGS

August 31-September 3 in Christchurch, New Zealand. The Third Congress of the Asia Pacific Association for Respiratory Care presents "Breathing into 2001," at the Christchurch Town Hall. Contact Stephanie K Humphries, The Planit Group Limited, Event Organizers, 201 Cambridge Terrace, Christchurch, New Zealand. (643) 366-5955, fax (643) 366-5944.

September 11-13 in Dallas, Texas. The Department of Health & Human Services, U.S. Public Health Service, and the Health Care Financing Administration announce the Second National Primary Care Conference, entitled "Making Primary Care Work Under Health Care Reform," at the Fairmont Hotel. The national forum focuses on changes under state health care reform as they relate to primary care systems, workforce issues, and needs of the underserved. Contact Rose Salton, Social and Scientific Systems, 7101 Wisconsin Ave, Suite 1300, Bethesda MD 20814. (301) 986-4870, fax (301) 913-0351.

September 26-27 in Ann Arbor, Michigan. The University of Michigan Medical School sponsors "Update on Pulmonary and Critical Care Medicine" at the University's Towsley Center. The course focuses on interstitial and inflammatory lung disorders, evolving technologies, and recent concepts in pulmonary medicine, critical care, and infectious diseases. Approved for 14 hours of AMA Category I credit of the American Medical Association's Physicians Recognition Award program. Write to Registrar, Towsley Center for Continuing Medical Education, Department of Postgraduate Medicine and Health Care Professions, University of Michigan Medical School, PO Box 1157, Ann Arbor MI 48106-1157.

Sept 29-Oct 2 in London, Ontario, Canada. The Respiratory Therapy Society of Ontario (RTSO) presents its 22nd Annual Education Forum at the Radisson Hotel. Contact the RTSO Head Office, 122 Cumberland St, Suite 200, Toronto, Ontario, Canada M5R 1A6. (800) 267-2687.

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C ❑ Anesthesia Service
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3 ❑ Critical Care
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7 ❑ Education
8 ❑ Management
IV Position
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C ❑ Supervisor
D ❑ Staff Technician
E ❑ Staff Therapist
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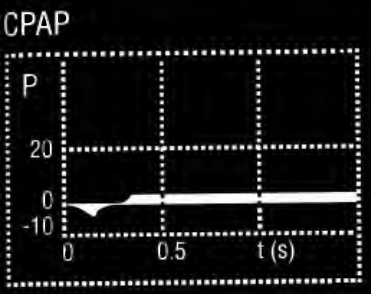

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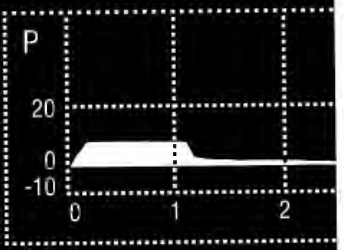

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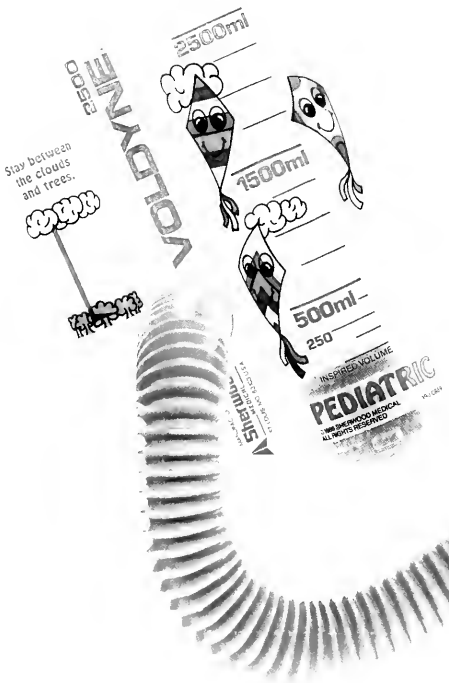
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